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THE PRACTITIONER

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THE
PRACTITIONER

A Medical Journal.

JANUARY-JUNE, 1907.

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149, *STRAND, LONDON, W.C.*
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THE PRACTITIONER.

JANUARY, 1907.

INFLUENZA.

By T. CLIFFORD ALLBUTT, M.D., LL.D., D.Sc., F.R.C.P., F.R.S.,

Regius Professor of Physic, University of Cambridge.

INTRODUCTION.

THE Editor of THE PRACTITIONER has requested me, and requested me so kindly, to write a few words introductory to the present collection of essays on the several aspects of influenza that, although I do not feel able to add much to the paper I had the honour of reading before the Hunterian Society in April, 1905, I feel still less able to forfeit the further honour of association with the remarkable company of observers and investigators which he has enlisted, even if it be only that of him who carries the wand and points to the diagrams. This task is the more difficult as influenza is of protean diseases the most protean; more diversified even than syphilis, with which infection it shares, may I say even still, the elusive behaviour of its infectious element. In both, this element is caught, as it were, by glimpses, and its action so largely modified by incidental conditions, that, in the one as in the other, diagnosis is often a delicate balance of probabilities. Dr. Newsholme brings out statistically the excessive pulmonary death-rate during epidemics of influenza, although in many individual cases it may be difficult to detect the specific cause with any certainty. He adds, only too suggestively, that were it as easy to deal by statistics with nervous and circulatory diseases as it is with the pulmonary, we should probably discover in these spheres of disease a like excessive prevalence. Well, then, may such a pervasive virus, fighting in part under its own flag, in part treacherously under other flags, be as disastrous at length, if not so appalling, as the plague itself. And not only is this subtle bane manifest directly and indirectly in its course, but, as more than one of the essayists tells us, it may feign, as it were, to pass away, only to return in some other guise. Almost as I write, a patient informs me that last spring he suffered obviously from influenza, and not lightly; then for three weeks at least the malady seemed to disappear, then came an attack of neuritis, which was worse in its disablement than the primary attack, and very much more protracted; here again its manners are like those of syphilis. The state of infection, says Dr. Newsholme, may persist for many months.

The bacteriological position of the problem is rather exasperating, as Dr. Emery tells us. Clinically regarded, influenza, in its well-marked cases, or in the characteristic sequels of less manifest attacks, is no indifferent or vague intoxication; the morbid series is as characteristic, let us say, as that of diphtheria, a disease which, through many diversities, preserves a substantial identity. Yet the responsible microbes seem to be several, as though two or more do, or in special circumstances may, distil a like or identical toxin; or, as though by the previous influence of one of them, the body may be so modified that a kindred microbe can effect upon it what it could not have effected alone. But for the discussion of these larger aspects of bacteriology, I am thankful to be able to refer the reader to the masterly paper which Dr. Andrewes has recently published.

The baneful effects of conspiracies of influenza with other infections is most salient in its association with the tubercle bacillus, and with the pneumococcus. Some such reinforcement we all perhaps have seen in local outbreaks of disease of ambiguous character, such as the "cerebrospinal" epidemic, described so well by Dunn and Gordon in Hertfordshire in 1905; and the epidemic of low and fatal pneumonia, which broke out in Saffron Walden this year, the cases of which, as I saw repeatedly for myself, depended on no uniformity of external conditions, but attacked individuals, as it were, capriciously. Thus "mixed infections" play a large part in the devastations of influenza; as in scarlet fever, the miasm takes with it other evil spirits even worse than itself; each giving to each an intensified virulence. How destructive mixed infections may be in local mischief is illustrated by the articles of Mr. Cheatle and Dr. Thomson. Again, influenza simulates other diseases; the essayists repeat how hard it is at times to distinguish influenza from typhoid fever. Dr. Dalton tells us that influenza is even capable of producing rose spots, and I can bear personal testimony that this is true. Dr. Savage has noted its conspiracy in acute syphilitic mania, and in the acceleration of general paralysis. In its fulminating forms, as in the person of one of my colleagues in Cambridge, the suggestion of acute meningitis was startling, for, even in this disease, I have not seen headache more agonising, or more distressing vomiting.

The equivocal bacteriology of influenza and the dubious significance of the *M. catarrhalis*, set clearly forth by Dr. Emery, are illustrated by Dr. StClair Thomson's instance of two nurses, attacked at the same time, in the one of whom was found the *M. catarrhalis*, in the other the pneumococcus. Dr. Horder's remark that, if the influenzal bacillus finds its way into the blood, its vulnerability, generally, though not always, leads to its destruction, is of great practical importance ; as well as his note that its successful entrance into the blood may be betrayed by a leucocytosis.

The infectiousness of influenza seems now so obvious that we find it difficult to realise that, in 1889–1892, this law was far indeed from general acceptance. Parkes denied infection from man to man ; and when I was brought into the great field of its operations in the asylums of England, a vast experiment which to the most careless looker on displayed personal contagion in huge proportions, it was with some reluctance that we divested ourselves of the atmospheric hypothesis. Yet to others the truth had been apparent ; Sir Peter Eade had tabulated the returns of public institutions in earlier epidemics to prove personal contagion ; and Dr. Bardsley, in 1803, showed similar results in the experience of the Manchester Lunatic Hospital. Dr. Dawson Williams has pointed out to me a report of the Medical Society of London on influenza, a report published in 1805 upon the testimony of circulars sent out by the Society in 1803 (*M. S. Tr.*, Vol. VI.) proving definitely from the events in isolated communities and individuals that personal infection played the chief if not the only part in the spread of the disease. That, as I said in 1905, it is in its respiratory forms by catarrhal expectoration and spray, that the infection is conveyed, seems to be unsupported by the experience of many trustworthy correspondents. There is some notion that it is while the temperature is raised that infectiousness is active; but perhaps none of these more exclusive axioms is adequate. I have also to modify my notion, then published, that immunity usually persists so long as six months; many cases have been brought to my notice where such an interval seemed improbable—where, I mean, the succeeding attack was probably not a relapse, but a new infection. I have myself also seen lately two attacks, apparently separate, to occur in the same person within two months.

As to symptoms, all will read with pleasure Sir John Moore's bright and clever sketch of the malady. Of its sudden onset I have two notable cases to add to those, with which I illustrated this characteristic feature in 1905. The first was sent me by Dr. Dawson Williams; it occurred in a gentleman who, when working actively in his garden, was called into tea; he turned to go in but, as he did so, was seized with a sense of prostration so utter that he could hardly crawl to the window, and he crossed its threshold only to sink exhausted into his armchair. The second, which was narrated to me by Dr. Cane, of Edmonton, was even more remarkable, as a feature of the onset was a sudden assault on the mind. This victim, when attacked, was at a railway station, in the full sense of well-being and happiness, when so sudden a stroke of misery befel him that he had to implore his companion to carry him into the waiting room, and to detain him there forcibly, lest he should seek destruction by throwing himself under a passing train. And, as I stated before, the misery may as suddenly vanish. Of such sudden recoveries, Sir William Church told me that after weeks of depression, and while going about his work in a doleful, perfunctory spirit, the patient bethought himself, almost in a moment, that afternoon tea would be very welcome, and promptly welcome it was; it tasted delicious, and the enjoyment of it was the prelude, or rather the fulfilment of release from his despondency. The loss of smell and taste, so definitely recognised in influenza, may have far-reaching effects. In a case, in which Dr. Sheehy, of Totteridge, was so good as to consult me, the chief distress of an attack of influenza was "an overmastering sense of not being in her right mind" ; this obsession suddenly disappeared on a certain day and hour, the date of which was definitely noted at the time. That the fever is erratic, intermittent, and irregular, and that it bears no proportion to the malignancy of the attack, is once more set forth in these pages. An eminent physiologist has told me that, in one of his attacks, the temperature was subnormal throughout, and this from the very beginning.

To the sweats, of which I spoke in my former paper, a sequel which, it appears, was not generally recognised, I have had many further witnesses. Dr. Beverley, of Norwich, was, I think, the first to suggest that the "sweating sickness" may have been a virulent influenza. To the sweats I may add recurring rigors with elevations of temperature lasting a day or two, and due perhaps to sallies of Dr. Emery's vulnerable bacilli in the blood. Such a case I saw more than once with Dr. Windsor, of Royston ; and, anxious as we were lest there should be latent suppuration, we hugged the belief that they would prove to be but the sequel of a recent influenza ; and so it proved ; after some half dozen of them they vanished, leaving no trace behind.

On the difficult subject of gastro-intestinal influenza, Professor Dalton's article is welcome. I hear that this form is just now prevailing in Oxford. During epidemics I think it does not present much difficulty of diagnosis. Occurring as sporadic cases, the difficulty may often be great, even, as Professor Dalton says, so great as to suggest abdominal operation. The curious separation of the gastric and intestinal phases, which he describes, might indicate the nature of the attack. Of atonic distension of the stomach, described by Sir William Broadbent and myself for many years past, influenza convalescence offers but too many examples.

As the muscular coat of the stomach, so suffers the muscle of the heart. It is unnecessary to dwell on the manifold and instant perils of the influenzal heart ; but it may not be out of place to advert once again to the terrible rapidity of the collapse of not a few elderly or weakly persons attacked by influenzal pneumonia, be it ever so moderate in manner, and the patient of no unfavourable an aspect for his years. After little or no previous warning, in an hour or two, as Sir William

Moore tells us, the tide may turn, and life may be swiftly ebbing away. Dr. James Mackenzie has demonstrated, by his graphic method, how the poison directly affects the cardiac muscle; and Dr. Horder, if I mistake not, has found the bacillus of Pfeiffer in this muscle. In too many cases, I have found mitral murmurs, audible in the back, to persist long after convalescence; and even without valvular disorder, dilatation of the right heart, or of both sides, may be obstinate and grievous. Two such cases I well remember, under the care of Dr. Michell, of Cambridge. It would seem that influenza may account for some cases of chronic septic endocarditis, but that in influenza, the mode is verrucose, not ulcerative.

Dr. Ford Anderson, who, with Dr. Parsons and Dr. Bulloch, contributed so effectively to the Medical Society's discussion in 1905, supports Dr. Maurice Craig's observation that, in cases of influenzal melancholia, he has found the arterial pressures excessive. I and others have noted that after influenza the arteries may be thickened. To the cases of influenzal angina pectoris narrated by Dr. Sansom and by myself (*Cavendish Lecture*, 1903) farther instances have been reported by Dr. Arthur Shadwell, Dr. Eames of Uffculme, and, in this issue, by Sir Douglas Powell and Dr. West.

In the essays on respiratory influenza much interesting matter is added, some points elucidated, some reinforced. The laryngeal spasmodic and paretic phenomena are described fully and convincingly. To the pneumonic phases I would add the following case:—A gentleman, often attacked by influenza, fell again a victim. About the third or fourth day, on careful examination of the chest, I found slight dulness over the posterior aspect of the left upper lobe, under the scapula. This patch, of doubtful interpretation at first, soon became definite enough, as an area of well-marked dulness of some three or four inches in diameter. It was watched with no little anxiety, but happily in a few more days it began to decrease in diameter, and, as compared with the consolidations of Dr. Hector Mackenzie's description, receded quickly; that is, in a week after it began to recede, there was but little of it remaining. To auscultation the phenomena, as in Dr. Mackenzie's experience, were ill marked, amounting to little more than altered quality of respiration; the quality, however, could not be called tubular, and added sounds were barely perceptible. The cough was not severe and the sputum was scanty. A specimen was examined at our laboratories, and reported as giving a pure culture of the influenza bacillus. It is in the pneumonias that the tragical heart failures already described are prone to occur. When influenza is about, the diagnosis of influenzal pneumonia, or of an influenzal element in a lobar pneumonia, is usually not difficult of appreciation ; the fever is fitful and out of proportion to the illness, and the differences of the sputum, the premature sweats, and the exhaustion are but too significant. I have still to report that excavation of the lung may occur in influenza without the tubercle bacillus ; fairly frequently with discovery of pure cultures of a microbe of the influenzal kindred. Empyemas of influenzal origin, in my experience, do badly.

Under the head of the nervous caprices of influenza, Dr. Harris gives us interesting information, much of it of definite value, some of rather equivocal value, as no one knows better, no doubt, than the author himself, whose duty it was to record all facts which could in any degree be relevant. Still, if under strict criticism, the evidence of consecutive connexion of certain of the morbid series attributed to influenza is in many cases considerable, in others, it is open to much doubt. Yet, as Dr. Savage and Dr. Claye Shaw testify, if certain graver affections of the nervous centres are not of influenzal origin, they may, by its interference, be grievously accelerated. I cannot admit that the neuropathic consequences of influenza generally require a neurotic stock on which to engraft themselves ; on the contrary, severe and prolonged as in these cases they may be, I have been surprised again and again to see them appear in no slight severity, and only too protracted, in persons of well balanced temper, steady health, and good family history. It is chiefly from the neuropathic class, no doubt, that the relatively incurable cases are derived which drift into asylums. In pains of the face, it is of the first importance, as Dr. StClair Thomson tells us, to take every precaution to ascertain the condition of the teeth (Head), the sinuses, the ears, etc., lest an operation be either unduly postponed, or performed without reason. That bulbar palsy may sometimes be purely influenzal seems revealed by transitory duration and happy issue. If the dangers to the respiratory centres and nerves are less than in diphtheria, they are not negligible, as I said in 1905, and have witnessed since in less grave degrees. On the other hand, how transient influenzal palsies may be, is illustrated by the following case I saw, some years ago, in an elderly man. Early one morning on rising from bed, he found his right hand palsied and hemiplegia seemed to be threatening; and indeed that some thrombotic progress was already at work in the brain seemed probable to me and to his family physician. However, after but little aggravation, up to the degree of disabling him from signing his name, the palsy set itself to recede; and, in two or three weeks, the hand and wrist were restored to their normal condition. On careful consideration, an attack of influenza a few days before was recognised, and the source of the infection also. No sign of palsy appeared at a later date.

How puzzling areas of neuritis may be in influenza is well shown by the following two cases recently under my observation. In one of them, the patient, a healthy man, was seriously disturbed by a most perplexing and uncomfortable anæsthesia of the left cheek, and lip or lips. Some weeks later I happened to fall into conversation with him on the annoyances and apprehensions he had endured, and, knowing that he was very susceptible to the disease, I opined that his trouble was influenzal, and would gradually disappear. And so it did. But not till a few months ago was the nature of the case put beyond doubt, and in this way; that the patient was attacked again, somewhat severely, by influenza, and, two or three weeks later, the anæsthesia of the cheek and lip returned, again to disappear slowly and harmlessly. A far greater alarm arose in a little boy, aged 5, in whom, after a day or two of scarcely recognised feverishness, ptosis of the left eye appeared slightly, and grew worse and worse for a day or two, till it became almost complete. Not only so, but the pupils became very unequal, one of them widely dilated and insensible to light. There was no headache, no vomiting, no great change of temper. Dr. David Lees saw the little boy with us, and, after careful review of the whole circumstances, and of the prevalence of influenza, decided against meningitis or diphtheria, and in favour of influenza. The palsies cleared up slowly but did not wholly disappear for some months. Dr. Lyle's article throws more light on this case.

Sir Peter Ede has described to me an "eczema-like eruption" which, in former years, he had observed in the mouth in influenza, and Dr. A. Bonny had noted it likewise. I must admit that I have not seen it, but also that I have not looked for it. If there is anything characteristic of the kind, it may be very useful in diagnosis; and an extension of it might account for some of the suppurations of the passages about the mouth and nose. The importance of minute attention to the state of the mouth, as to cleanliness and moisture, in all the infectious fevers, is receiving the scrutiny it deserves.

As psychical attacks may be likewise as transient as they are alarming, Dr. Claye Shawe warns us, if influenza is in question, to be slow to place the patient under certificates.

Finally, as to treatment. Even at the beginning there must be no tampering with depressing drugs or regimen. Our essayists advise us to avoid even the salicylates, though salicin, or, in case of myalgia, aspirin, may be administered, and the aspirin may be guarded by caffeine. There is no doubt that promptly to betake himself to bed is the first duty of the patient, both for immediate relief and for the aversion of evil consequences. And so long as active symptoms remain, the rest in bed must be insisted upon. To go abroad before the poison is fully eliminated, is to run a risk of grave complications. Dr. Hector Mackenzie's plea for fresh air will receive a ready hearing. Sir William Broadbent's confidence in quinine is shared by many of us; and his remarkable testimony in these pages to its preventive value is of great weight and practical importance. It seems that sprays are of little service, even for the prevention of infection. For the fever, no cold baths are to be thought of, even cool spongings are ill borne. Indeed the fever is practically never itself menacing, nor requires special remedial means. Oil of cinnamon has been strongly recommended by Dr. Carne Ross, and deserves a trial, as Dr. Caiger and other physicians of special experience in infectious fevers are disposed to regard this drug with some favour. The dose is 10 m. every two hours at first, the frequency being reduced as circumstances indicate. I do not often see cases of influenza during the first days of the disease and have no opinion as to its efficacy. In the prostration of pneumonia, Dr. Hector Mackenzie testifies still to the virtue of alcohol, and I must declare that I agree with him. For the sweats, which may continue, or even set in, long after apparent convalescence, I am clear that the ordinary means of restoring tone to the skin are at best of little use, and they are disagreeable, if not injurious, to the patient. For this distressing consequence, cool spongings, douches, massage, and the like are to be avoided, even the change of the clothes must be done carefully, the fresh ones being warmed before use. The surface of the body must be dried gently with a soft woollen towel. In the case under the care of Dr. Sheehy already referred to, Dr. Sheehy discovered almost accidentally, certainly contrary to our expectation, that the sweats were controlled and cured by the nitrites. The form used in this case was amyl nitrite, of which $\frac{1}{10}$th of a minim was administered on sugar, three or four times a day, a dose which, of course, may, if necessary, be increased.[1] Massage, improper, of course, during the acute phases of the disease, is occasionally efficient in the later; but, generally speaking, it has not the success which might have been expected; perhaps it is too fatiguing for a profoundly debilitating disease. If tried during convalescence or for some sequel, it must be performed by a really skilful hand: there is much massage abroad, of which I wish that the worst that could be said were that it is perfunctory; much of it, by chafing and pounding, positively jading. If the instructions in Dr. Playfair's book were more sedulously observed in the training of massors, massagers, or whatsoever we are to call them in English, we should attain far better results.

No one knows better than the writer the degree of indulgence needed for these fragmentary remarks upon the following essays by men far better able than himself to deal with the subject.

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[1] Dr. West's papers, in which the same remedy proved effectual, did not reach me till my remarks had gone to press. Nor had I seen that of Sir R. Douglas Powell.

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A NOTE ON SOME CLINICAL ASPECTS OF INFLUENZA.

By SIR RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D.,
President of the Royal College of Physicians; Physician Extraordinary to His Majesty the King.

I really have nothing that is of novel interest to say about influenza. Its symptomatology has been more than adequately expounded although perhaps we still rather lack a due order and perspective in the formal accounts we read of this multifarious malady. I take it, however, that you want rather memoranda that may one day be worked into a good and ordered text-book account of the disease.

The catarrhal form of influenza as affecting the lungs is attended with physical signs which are absolutely characteristic of the disease, viz., the explosive inspiratory crepitus scattered through the lungs, especially the posterior lobes, often wandering from one part of the lung to another unattended with percussion dulness; associated with a straining paroxysmal cough at first with little and very difficult expectoration, which in a few days becomes very profuse, glutinous, then purulent. Many weeks before the recognition of influenza in the epidemic of 1890, I saw sporadic cases, in which with cough and fever, these peculiar physical signs were present which I thought must be occasioned by a kind of inflammatory œdema around scattered miliary tubercles, but they all got well. I had never seen anything like these cases before, but in the great epidemic which followed they were common enough. Of course, with pneumococcal infection superadded, the broncho-pulmonary catarrhal signs are merged in those of an ordinary pneumonia which differs from simple pneumonia in the increased tendency to paralytic filling of the lungs and cardiac failure. I should say that the cases of pneumonia in influenza are always due to pneumococcal complications, if I may judge from several consecutive cases in which the pneumococcus was invariably found. In the middle years of the epidemic more than at first and latterly I used to see cases, in which towards the decline of the attack acute anginal heart seizures occurred. There was no cardiac lesion, there had not necessarily been during the attack any special strain upon the heart, the seizures were severe anginal cramps of the heart attended with small thready pulse which occurred in quite young as well as in older people. I cannot recall any cases of this type that were actually fatal, and, although threatening enough, I felt able to give a good prognosis with some reserves with regard to the older people. There is one point with regard to these cases, which I have not seen alluded to, and which, although not special to them, yet I came to think accounted for the anginal phenomena by way of increasing arterial tension, viz., that the urine towards the close of the acute attack of so far uncomplicated influenza in these cases particularly was of high specific gravity, and with such an excess of urea as to become solid with deposition of scaly nitrate crystals on the addition of nitric acid.

Of nerve phenomena, besides the intense headache, severe depression (sometimes with mental aberration resembling that which may follow enteric fever, but more remarkable as a sequel to so short an illness as influenza) and sleeplessness, I, like others, have seen a few cases of paralysis, paraplegia, and, in one case, affecting all the limbs, of apparently the same type as diphtheritic paralysis, only not involving the fauces. I have seen one case of fatal neuritis attended with intense hyperæsthesia, which I believed to be influenzal, and I have recently heard of a fatal case in which the diaphragm became involved.

The prevalence of neuritis, since the commencement of the 1890 epidemic, is within the experience of most of us. A remarkable neurosis is evinced in the occasional occurrence of true spasmodic asthma, as a sequel to influenza, in middle-aged people, with no predisposition, and who have never before suffered from that malady. Many such cases have come under my notice. The majority of these recover, and they are cases in which balmo-therapeutics are valuable, but I have seen some very prolonged cases running the usual intermittent course of asthma. I should be inclined to class these cases with those of spurious angina, to which I have already alluded. It is clear that they are not dependent upon any coarse lesion of the nerve centres; they certainly are met with in persons of gouty diathesis.

NOTE ON THE THERAPEUTICS AND PROPHYLAXIS OF INFLUENZA BY QUININE.

By SIR WILLIAM BROADBENT, Bart., K.C.V.O., M.D., F.R.C.P., F R.S.,
Physician in Ordinary to His Majesty the King.

FROM the time of the first invasion of influenza, I have found that the best remedy was quinine. My usual prescription was one drachm of ammoniated quinine and two drachms of liquor ammoniæ acetatis every hour for three hours and then every four hours. In the fulminating attacks of influenza, in which the patient has become comatose, hydrobromate of quinine, given hypodermically in large doses, has completely relieved the unconsciousness.

As a prophylactic, I early ordered two grains of quinine every morning during the prevalence of the epidemic and the results appeared to be good. Of course, patients who were taking quinine, did occasionally get influenza, but I have known very many instances in which this dose has made a complete difference in the patient's liability to infection, and even in the general mode of life.

I have, moreover, had opportunities of obtaining extraordinary evidence of its protective power. In a large public school, it was ordered to be taken every morning. Some of the boys in the school were home boarders, and it was found that, while the boarders at the school took the quinine in the presence of a master every morning, there were scarcely any cases of influenza among them, although the home boarders suffered nearly as much as before.

In a large girls' school near London, the same thing was ordered, and the girls and mistresses took their morning dose but the servants were forgotten. The result was that scarcely any girl or mistress suffered, while the servants were all down with influenza.

A NOTE ON A POSSIBLE SOURCE OF INFLUENZA.

By **SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,**
Consulting Physician to St. Bartholomew's Hospital, &c.

One point in relation to the prevalence of influenza, during the last ten years, has been the undue frequency of the disease amongst visitors to the European Continent, especially to Paris and the Mediterranean littoral. People in perfect health have been struck down after a very brief stay in various places, or after making a rapid journey to the South. It has frequently occurred to me that they must have been infected in their sleeping apartments, or sleeping berths in the trains. Of course there is no evidence in support of this view, but it is not difficult to imagine that, in imperfectly ventilated rooms, and the compartments of the ordinary wagon-lits, there may linger for a long period the specific germs of this malady, so well termed by our American kinsfolk "the mean disease." We are painfully familiar with the fact that our neighbours on the other side of La Manche have an inbred dread of such ventilation as is deemed necessary for Britons, and revel all night in foul air and stuffiness, in the vain hope of avoiding catarrhs.

I am thus emboldened to believe that some effective measures for disinfecting or renewing the furniture, curtains, and blankets in such rooms, or sleeping cars, as are frequently used on the main routes of travel, would materially diminish the risks in question.

In any case, I am of opinion that this matter is well worthy of attention and inquiry, for it is a serious addition to the burden of frail and weakly invalids, who are seeking better climates, to be laid low with influenza and its many far-reaching sequels. Such an attack quickly curtails the benefits and refreshment naturally sought by persons who start out in fair health, and with high hopes of escaping from our sunless environment for a well-earned holiday in winter.

THOUGHTS ON INFLUENZA.

BY SIR JOHN MOORE, M.D., D.P.H., DUBL.; HONORARY D.Sc., OXON., F.R.C.P.I.,

Physician to the Meath Hospital, Dublin; and Professor of Medicine in the Royal College of Surgeons in Ireland.

WHEN I was a boy the term "influenza" was in constant use to describe an attack of acute catarrh of the respiratory mucous membranes from the nose to the bronchial tubes. It was, as it were, an echo of the great pandemic of influenza, which raged in the winter of 1847–1848. As years rolled by, the name fell more and more into disuse, until "influenza" at last became a mere tradition in medical nomenclature.

And so things continued until the year 1889, when, with all the suddenness of a volcanic eruption, or of a devastating conflagration, the disease once more spread over the habitable globe. In May of the year named, influenza appeared in Bokhara, in Central Asia. Thence it travelled north-eastwards to Tomsk in Siberia, north-westwards to St. Petersburg, westwards to the Caucasus and Southern Russia. In the last quarter of the same year, the epidemic, soon to become a pandemic, spread rapidly from Eastern Europe to the central and western countries of this Continent. Early in 1890, it overran America, Africa, and Australia, so that, in the words of its historian, Ditmar Finkler, of Bonn, "the high flood of the pandemic flowed over the whole globe in the space of a few months."[1] To return to the metaphor of a conflagration, the historic pandemic of 1889–1890 was followed by repeated "flarings" during the succeeding decade and a half, and, even to the present day, the tradition of the great outbreak finds expression in a diagnosis of influenza on very slender clinical grounds, and in a passionate fondness for the term on the part of a startled public.

In illustration of the truth of this statement, I may quote a letter received, so recently as October 30th, 1906, from a very popular and clever medical practitioner in Dublin. He wrote to me as follows:—"The bearer, Mr. A. B., is suffering from catarrhal influenza. He is anxious to go under your care into the Meath Hospital. Kindly admit him." When the patient presented himself in my consulting room in the evening, he had a coated tongue, and a rapid pulse, felt chilly, with a mouth temperature of 99° F.; his conjunctivæ were injected and there was a great deal of tremor about his hands and in his tongue. There was a vinous smell from his breath. I taxed him with drinking, and he admitted that he had spent the day imbibing brandy as an antidote to influenza. He undoubtedly had a coryza, and I admitted him to hospital, although I was sceptical as to the diagnosis of influenza. Next morning, on examining the patient, I found the liver enlarged and tender on pressure. Questioning the young man, he was seven and twenty, I ascertained that he had until recently been a grocer's assistant. On cross-examination he admitted that he had been in the habit of taking much more than was good for him, and I have a shrewd suspicion that this was the reason of his leaving the establishment in which he had been employed. He was, no doubt, the subject of "influenzal" catarrh in a certain sense, but the "influence" was alcoholic.

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[1] *Twentieth Century Practice of Medicine*, Vol. XV., p. 43.

My idea of influenza is embodied in the following definition in an article which I wrote for the *Encyclopædia Medica*, in 1901:—

"Influenza is an acute, specific, infective febrile affection, characterised by its remarkably sudden onset, after an incubation of about two or three days, the many symptomatic phases it presents, the singular predisposition to other secondary infections which it induces, its disastrous effect upon the heart, and the protracted convalescence which follows in its wake. In itself by no means a mortal disease, influenza exercises a far-reaching and malignant effect upon the death-rate, which surges far above the average during the few days or weeks of its epidemic or pandemic prevalence. Catarrhal symptoms, although often present, are not an essential feature of the disease. The victims of influenza are adults, who perish from pneumonia or bronchitis; and the aged, who sink from heart exhaustion. Children, while prone to the disease, enjoy a comparative immunity from its complications and dangers; in them a sharp febrile movement, with or without an evanescent rash of an erythematous nature, constitutes the main feature of the attack. Unlike the poison of typhus, the virus of influenza is not rendered inert by oxidation from contact with atmospheric air, but rather the contrary."

The virus, or contagium, of influenza, when once introduced into the system, acts, in the first instance, on the nervous system, quickly producing the phenomena of an acute pyrexia, with singularly rapid pulse. In January 1890, a boy of seven and a half years had incessant nausea and vomiting for 24 hours, and profuse sweating, with a pulse as fast as in scarlet fever, 140 per minute. At the same time, very young children seem to enjoy a comparative immunity from the disease, or to have it in a mild form, an ephemeral fever, followed by profuse sweating, and, after a few days, slight catarrh. In 1762, Sir George Baker wrote, *Leviter plectebantur infantes et liberabantur facillime.* This "child-type" of influenza was very familiar to me in a large girls' school in 1890.

In the article in the *Encyclopædia Medica*, I mention that adults, in most instances, suffer much more severely than children from the influenza poison. In them the attack, as a rule, comes on with lightning-like speed. A person apparently in perfect health is suddenly overcome by a feeling of general discomfort and profound depression. He feels chilly, or is shaken with a rigor worthy of an ague fit. His head aches. There are pains in his eyeballs, or "behind the eyes," and the eyeballs are exquisitely tender on pressure. Soon rheumatoid pains rack his body. They are particularly severe in the nape of the neck, small of the back, in the knees, and along the margins of the ribs. The patient is often sleepless, sometimes delirious. He may temporarily suffer loss of the special senses of smell, taste, and sometimes hearing. His eyes smart, tears overflow, he shrinks from the light (photophobia). Occasionally there is intense earache (otalgia). Strength ebbs away far more quickly than even in typhus, so that, after a few hours, he must take to his bed. Nor do the digestive and respiratory systems escape. The tongue becomes thickly coated with a creamy or blanket-like fur. There is a bad taste in the mouth, the breath is heavy or even fœtid. There is complete loss of appetite, amounting to a loathing against food. Nausea, and perhaps vomiting, add to the sufferer's distress. The bowels are constipated, but occasionally diarrhœa sets in, as in an attack of cholerine. In another group of cases, the brunt of the poison appears to fall on the respiratory tract—cough, expectoration, a sense of oppression on the chest, a tightness in the breathing, being common symptoms. In a word, influenza seems to have the property of picking out the weak point in a person's constitution. If the patient is neurotic, nervous and neuralgic symptoms are likely to develop. Any old tendency to catarrh of either the respiratory or digestive mucous membrane, is at once intensified in the presence or in the wake of this strange malady. At the same time, catarrhal symptoms may be badly marked or entirely absent.

The protean forms which influenza presents, from a clinical point of view, arise from the aptitude the poison has for fastening on the centres of least resistance in the constitution. Of the many types described perhaps these stand out most prominently :—

1. The neurotic, neuralgic, or rheumatoid type ;

2. The cardio-pulmonary type, in which the ebbing of the strength in elderly people is sometimes awful, often absolutely beyond control ;

3. The gastric or gastro-intestinal type, in which anorexia is present, sometimes to the extent of a loathing for all food ;

4. The febrile type, which prevails especially among children. The febrile movement usually lasts two or three days, and is succeeded by marked subnormal temperatures, so constantly present as to become an important diagnostic sign.

Influenza shows a decided tendency to relapse, a feature to which the indirect fatality of the disease is in great measure due. So far from establishing immunity, an attack of this malady seems to render an individual more liable to contract the disease upon any future exposure to its contagion.

In estimating the mortality of influenza, we must remember how vast are the numbers of the attacked. The death-rate is probably not more than 2 per cent. of all the cases. Next to the heart, the lungs are the seat of fatal complications. Influenza *poisons* the heart, and it is interesting to note that the phrase "heart failure," now on everyone's lips, was scarcely ever heard before the fatal pandemic of 1889 and 1890. The very treacherous and dangerous form of influenza, known as the cardiac type, may occur with or without pneumonia. In some cases, uncomplicated with pneumonia, the heart collapses, it may be with a suddenness that suggests rupture or cardiac thrombosis. Or again, the frightful ebbing of strength, in many cases of influenzal pneumonia, in persons of middle life or advanced in years, reaches its climax in a true heart palsy.

Pneumonia.—Foremost among the complications, to which the victims of influenza pay the heaviest death-toll, stands pneumonia, a term which, in this instance, must be taken in a generic rather than in a specific sense. Pfeiffer's *Bacillus influenzæ* may itself produce a primary pneumonia, and lobular rather than lobar in its distribution. Or, again, influenza may enormously increase the susceptibility of an individual to infection by Fränkel's *Diplococcus pneumoniæ*, in which case a true pneumonic fever may be indicated by its lobar distribution and by its croupous or, preferably, fibrinous exudation. In a third group of cases, a bronchial catarrh of true influenzal origin may travel into the capillary tubes, giving rise to a dangerous bronchiolitis and broncho-pneumonia. These are all well-recognised varieties of lung-inflammation which occur in or after influenza.

Nothing is more remarkable about these influenzal pneumonias than their insidious onset and treacherous atypical course.

Influenza is a perilous complication of pulmonary consumption. In London, at the height of the epidemic of 1889–1890, the weekly number of deaths from phthisis rose to double the average. In Dublin, during the first quarter of 1890, the mortality from phthisis was 26 per cent. in excess of the average for the corresponding period of the previous ten years.

Turning to the nervous system, I am in a position to corroborate Finkler's statement that "there is hardly a nervous symptom in existence which has not been observed in influenza." For this, he believes that we must seek an explanation in the action of some soluble toxin. But I cannot agree with that authority in looking upon all these nervous symptoms as purely functional disturbances. Organic lesions of the nervous system are by no means unknown in connection with influenza. For this statement there is abundant evidence in the literature.

Apart altogether from disturbances of the brain, spinal cord, and peripheral nerves, whether functional or organic, the disturbances of the mind, or psychoses, which attend or follow this strange malady, are countless in number and in variety. And, in addition to graver diseases of the mind, we must note the not infrequent occurrence of either hysteria, or hypochondriasis, in the later stages of influenza. The mental state of the convalescent is often intensely subjective or self-conscious.

In the article on "Influenza," in the *Encyclopædia Medica* from which I have already so freely quoted, I point out that the treatment of this affection is based on common-sense principles. It is expectant, palliative, symptomatic. There is no specific for influenza ; nor, up to the present, are there means at our disposal for securing immunity against its infection. The susceptibility to the disease varies remarkably from time to time in the same individual. One attack confers no immunity, rather the reverse.

The *prophylaxis* of the malady turns on strict isolation. Attention should be paid to the "toilet" of the mouth and fauces. When a person is struck by influenza, only one course lies open, and that is, to take to bed with the least possible delay. Rest, warmth, and quiet are three sovereign remedies for the primary disease, and the best preventives of its secondary complications.

Probably the most popular remedy is ammoniated tincture of quinine. Its disagreeable taste is palliated by the addition to each drachm of the same quantity of syrup of orange peel. A dessertspoonful of this combination, equivalent to 1 grain of quinine, should be taken in water every fourth, sixth, or eighth hour.

To relieve the distressing rheumatoid, or neuralgic pains of influenza, salicylate of sodium may be given in combination with granular effervescent citrate of caffein. But the most useful combinations for this purpose are phenazone (3 to 5 grains for a dose), tincture of gelsemium (5 minims), and chloroform water ($\frac{1}{2}$ an oz.); or acetanilide (7 grains), caffein (1 grain), and sodium bicarbonate (2 grains), in a cachet.

Either of these prescriptions may be repeated every two or three hours till pain subsides. Phenazone, even in small doses, is unsuitable for children; for them, phenacetin is safer as a sedative and analgesic. Be it remembered that these remedies should be given to relieve pain, not pushed so as to reduce bodily temperature, which is a practice fraught with danger to the patient's life.

In "heart failure"—to use a popular phrase—and in the tedious convalescence which so often follows in influenza, strychnine is our sheet-anchor. In failing heart, it is best given hypodermically to the amount of gr. $\frac{1}{30}$ in combination with digitalin (gr. $\frac{1}{100}$), or with morphin, if the patient is restless or sleepless. Caffein, strophanthus, and "camphor elixir" are also useful remedies when the heart flags.

The physician's skill and tact will be tried to the uttermost in arranging the diet of an influenza patient, especially in the case of one suffering from the gastric type of the malady. But let him not despair, for the trouble he takes will meet its reward.

A word of warning in conclusion. The prescribing of alcoholic stimulants in influenza demands more than ordinary circumspection. The mental state in this malady is so excitable, so unstable, so impressionable—in a word, so neurotic, that the seeds of intemperance may be sown by following the unthinking advice to take wine or spirits as a stay in weakness. My conviction is that alcoholic stimulants are, generally, not only unnecessary but positively harmful in the treatment of influenza. If ordered at all, they should be given with, or in, food, and only for a limited time like any other powerful drug.

SOME GENERAL CONSIDERATIONS ON INFLUENZA.

By SAMUEL WEST, M.D., F.R.C.P.,
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THE present epidemic of influenza has lasted 16 years, a duration which appears to be unprecedented. The old accounts describe influenza as bursting upon a country like a sudden storm, which, after raging for a short time with great violence, disappeared entirely. This was so far true of the last epidemic in 1847–8. So it was that, when this attack broke out, no one then in practice could have had any acquaintance with the disease, unless he had been in practice in 1847–8; in other words, unless he was at least 65 years of age. Indeed, during that interval, the very conception of the disease had changed, so that what was then called influenza was the common cold in the head, or feverish catarrh.

Opinions, too, have changed in respect of its infectiousness. In former epidemics, and especially in that of 1847–8, the general conclusion was that influenza was not an infectious disease, *i.e.*, that it did not spread directly from the sick to the healthy, but that its prevalence depended upon some peculiar external conditions, called the epidemic influence, which accidentally developed, and rapidly disappeared.

Now influenza is regarded as eminently infectious. How far the general belief in its infectivity has been affected by the discovery of the influenza-bacillus, and by the assumption that all germ diseases must be infectious, it is not necessary to inquire. Still, assuming it to be very infectious, there are many peculiarities which call for explanation. How is it, for instance, that one member of a household may be picked out and the others escape, though they are susceptible, as is shown by their acquiring the disease shortly after in some other way? According to present notions, this can only be explained by the resistance of the individual varying greatly at different times, and being easily modified by accidental conditions. One of these modifying influences is, no doubt, exposure to cold.

Take, for instance, the case of two of the wards of St. Bartholomew's Hospital during the original outbreak. These wards remained entirely free for a considerable time, although the epidemic was raging violently outside, and the house physicians had to do their work in the wards while themselves suffering from the disease ; for, ill as they were, there was no one available to take their places. In spite of this, no one in the wards succumbed, until two convalescent patients were allowed to go to the examination hall. They got a chill, came back into the ward, and developed influenza the next day. Still, it did not spread further in the wards at that time.

From our present experience, we must conclude that influenza is infectious in a very high degree indeed, and that the protection afforded by an attack is imperfect, or of very short duration. Indeed, one attack seems actually to predispose, after a time, to another, or, to put it differently, that the positive phase of protection is followed by a negative phase, in which the individual seems rather more than less liable to succumb to infection if exposed to it. It seems more likely that an individual may never have influenza at all than that, having had it once, he should never have it again. Some, indeed, seem to offer so little resistance that they develop it regularly once or twice a year.

It is no doubt true that many of the attacks, which are now in a loose way diagnosed as influenza, are something else. Long before influenza came, in 1890, febrile attacks of indefinite character were frequent enough, which were called by various names, the commonest being febricula, or simple fever. Some of these were no doubt mild typhoid fever, which would be recognised now by the character of the temperature chart, and by Widal's reaction. Others were due to milk or food infection. Others what we now regard as feverish catarrh. Making due allowance for cases which are confused with influenza, there can be no doubt that true influenza has been more or less constantly with us for the last 16 years, and breaking out at intervals of every few months in fresh epidemics, until one may fairly begin to wonder if it will ever again leave us completely.

The great fact of the last 50 years, and which must be taken into account in considering infection, is the enormous increase in the facility of communication between the different parts of the world and of the same country. Formerly, when communication was difficult, the original attack conferred immunity for long enough time, and, no fresh infection being subsequently introduced, the disease disappeared. If, however, after a time, for the protection as stated must be of short duration, infection were reintroduced, as the facilities of communication render only too likely, it would find some individuals susceptible, and another outbreak would occur, though of more limited extent than the previous one.

So we should expect recrudescence under present conditions, and the complete disappearance of the disease would be rendered very difficult.

The Fever.—The characteristic fever of influenza is of short duration and of moderate intensity. The temperature rises suddenly, remains raised for two to three days, and falls fairly rapidly, though not with any characteristic crisis. In these respects, the cases differ widely. The temperature may be little, if at all raised, or be unusually high, and these have been described as the apyretic and hyperpyretic forms, but the distinction has no particularly practical importance. The change in the character of the fever, which seems to be taking place, is a tendency towards longer duration and less severity. This introduces fresh difficulties of diagnosis. Thus, if the fever is moderate and of eight to ten days' duration, the diagnosis of typhoid fever might arise, and if there were much headache, even of meningitis, or, if the fever were high and chest symptoms present, pneumonia might be suspected.

Post-febrile Depression.—Perhaps the most remarkable characteristic of influenza is the post-febrile depression, which may last for ten days or a fortnight after the fever is over, during which period the temperature is subnormal, the pulse and respiration slow, and the bodily powers greatly reduced. There are no other affections in which this stage is so marked except diphtheria, and typhoid fever.

This depression does not vary necessarily with the severity of the original attack, for it may be extreme after what appeared to be quite a mild attack, and some of the severer attacks do not show it much. It is of great importance, for it is during this period when all the vital powers are low, that complications are so likely to arise—complications which can be prevented by care and precipitated by neglect. So long as the temperature remains subnormal, which may be for 10 to 14 days, the resistance of the patient is reduced to its minimum. It is a good practical rule to permit no patient to return to work, or to run the risk of chill, until this stage is past, as will be shown by the rise of the temperature to the normal again.

During this stage the patient feels ill, good for nothing, and is often greatly depressed mentally. It is also very curious to note how suddenly, sometimes, these feelings of depression may pass away—as it were, almost by a crisis. One case will do as an illustration.

A gentleman, after a very mild attack, which was not severe enough to require a doctor, suffered for a month from the most painful depression ; all interest in life was gone, and he hardly seemed to care to live ; he was for the time profoundly melancholic. After a month, quite suddenly, at 12 o'clock in the day, he used to say, a cloud seemed suddenly to lift off him, and he exclaimed all at once, "I am well again," and all his depression vanished. On the other hand, this depression may last for months, and even end in melancholia.

In connection with this depression—physical and mental—may be mentioned a curious association, which I have several times observed, but which I do not remember to have seen recorded, the frequency with which this depression merges in the cachexia of new growth. Thus, I have seen patients in middle life, who had been in good health till the attack of influenza, but who were never well after. After a long depression stage, they became more and more thin and anæmic, and finally developed cancer, of which they died. I have met with such cases too frequently to be able to regard it as a mere accidental coincidence, and suppose that it must be explained on the assumption that influenza had greatly reduced the resistance of the body, so that it fell an easy prey to new growth.

Pneumonia.—The most common, and at the same time the most grave, complication of influenza is *pneumonia,* although this can hardly be rightly called a complication, for it usually develops a week or ten days after the attack of influenza is past, during the post-febrile depression stage.

It appears to be generally pneumococcal in nature, and not due to the influenza-bacillus, and it runs the ordinary pneumococcal course, beginning and ending suddenly in the usual acute way. But the prognosis is worse, occurring as it does in a patient already in a state of debility and depression.

Post-influenzal pneumonia often, however, presents peculiar features. It may begin in a much less sudden way, and end, not by crisis, but by a gradual fall, *i.e.*, by lysis, extending, it may be, over many days.

Its most remarkable character is the tendency it shows to creep or spread, so that it may be invading one part of the lung while resolving in another.

I have seen it spread thus from the base of the right lung to the apex, and then begin at the base of the left lung. This patient lived three weeks, and then died rather from exhaustion than suffocation.

Influenza-pneumonia may also develop in two or more different parts of the lung at the same time. So that, instead of being massive or lobar, it may be patchy and multiple and resemble bronchopneumonia. It is a good illustration of the view which I have long held, viz., that pneumococcal pneumonia may occur in either the lobar or the disseminated form.

One class of the kind deserves especial mention, because of its resemblance to phthisis. Here, with somewhat indefinite signs of patchy consolidation of the lung, the fever may continue with a marked evening rise, lasting, it may be, for some weeks. The patient may look and feel very ill, and become cachectic enough to suggest tuberculosis, to which the resemblance becomes still closer, if, as occasionally happens, there is some hæmoptysis. There are, however, no definite signs of tuberculosis at the time, or subsequently, and recovery, though delayed, is generally complete.

One case of this kind will serve as an illustration. A young healthy, and active man of 35 was attacked with influenza. He never picked up satisfactorily, the temperature began to rise, and reached 102 103 in the evening. A slight cough set in, and shortly after there were one or two slight attacks of hæmoptysis. The diagnosis had been made of phthisis, but I was led to take a less unfavourable view, and sent him abroad. After three months' holiday he returned well, and has continued so since (15 years). These cases of hæmoptysis, simulating phthisis, are recorded in the accounts of the old epidemics. They must have been very rare in the present epidemic ; at any rate they have not been often described.

Influenza stands in no special relation to phthisis. So far as can be judged, it is rare for a patient, as the result of influenza, to become phthisical, likely as it might be thought *a priori*—but, though influenza greatly reduces the resistance of the body to the pneumococcus, it does not seem to do so in any special way to the tubercle bacillus.

In a patient, already tubercular, however, influenza may easily excite a fresh outbreak, and lead to a recrudescence of the disease, which may previously have been for years quiescent.

The Heart.—I do not know of any case in which valvular disease could be fairly attributed to influenza, though it is a possibility, for Dr. Horder has recently published some cases of malignant endocarditis, in which the influenza bacillus was discovered in the blood during life, and, in the vegetations on the valves, post-mortem.

As cardiac attacks are not uncommon in the acute stage, and cardiac debility and failure frequently follow, it is clear that influenza has a profound effect upon the cardiac muscles and nerves. Indeed, in this respect, influenza bears a close resemblance to diphtheria.

The cardiac form of influenza presents two prominent symptoms *præcordine pain* or *cardiac syncope*, both occurring in paroxysms repeated, it may be, at short intervals.

Præcordial pain is of two kinds. In one, it is *neuralgic*, and not attended with any obvious cardiac symptoms ; in the other, it is *anginal*, and may actually be angina pectoris. This is associated with very grave cardiac symptoms.

The *neuralgic form* is limited to the præcordium and immediate neighbourhood, and does not radiate like angina to other parts. It occurs in intense paroxysms, so severe as to cause even a strong man to cry out. I have heard the sensation described as if a tiger lay with its paw upon the chest, and every now and then gave a grip with its claws driving them deep into the flesh. In their excruciating sharp paroxysmal character, these influenza-pains can only be compared with the lightning-pains of locomotor ataxy. Beyond the agony they cause, they are not of any graver importance than are the similar neuralgic pains which occur in other parts of the body.

The *anginal form*, on the other hand, is of very grave import. It is often of the same character as true angina, radiates in a similar way, and is associated with signs of acute cardiac syncope. These attacks are very alarming, and often prove fatal. Attacks of cardiac syncope are not necessarily associated with paroxysms of anginal pain. There may indeed be no præcordial sensation at all, or, at the most, an uncomfortable feeling of tightness, or oppression. The attacks may come on like faints, suddenly, without any warning, or apparent cause. The heart's action then becomes suddenly very feeble and irregular, and the sounds almost inaudible. The pulse at the wrist is hardly to be felt, and the respirations are slow and feeble, as if the patient were too weak and weary even to breathe. He looks extremely pallid, and as if about to die, as indeed he may if not assisted at once by ether and strychnine either by the mouth, or subcutaneously. The attacks may be of every degree of severity. The slighter ones resemble, if they are not actually the same, those attacks of sinking over the heart and epigastrium with faintness, in which the patient complains of feeling as if he were sinking through the floor, and calls out urgently for stimulants or food to relieve them.

In the epidemic of 1847, cardiac attacks seem to have been very common, but in this epidemic, though occasionally seen, they have been comparatively rare.

A good instance of these heart attacks and their course is given by the following case:—

A young man, of 30, strong, big, and muscular, who, on several occasions, for about seven to ten days, had attacks of the kind, in which he seemed about to die, and probably would have died had not the doctor stayed with him for hours and kept some competent person near to administer the suitable injections and medicines when occasion arose. In many of the attacks, death seemed imminent. Recovery appeared almost miraculous, and was entirely due to the devotion of the doctor and nurse, for nothing else could have saved his life.

As would be expected, the heart remained very weak for a long time after the acute stage had passed. For many months he could not walk without shortness of breath and palpitation, gradually the symptoms improved, but, for about a year, he was practically a cardiac cripple. Ultimately he completely recovered, and is now, 14 years later, strong, active, and free from all signs of cardiac trouble, though he has lived a strenuous life.

This was the most serious case of the kind I ever saw, but it shows that, even in the gravest form, no organic lesion may be left which would render complete recovery impossible. Recovery in this case was largely due no doubt to the age, for had he been 20 or 30 years older, it is pretty certain that he would have died.

Cardiac weakness may be left behind by influenza, even when there has been no cardiac symptom at the time of the attack. This is seen in old and young alike, but is, of course, of more serious import as the patient is getting on in years. I suppose in all these cases that the effect is due to the action of the influenza toxines upon the heart-muscle or nerves, as it is in diphtheria.

The prognosis of post-influenza cardiac debility is on the whole good. Recovery in the end practically always occurs in the young, although in the elderly it may be delayed, and in the aged may fail.

Sweating.—One other peculiarity may be mentioned. In some cases, especially in women, the sweating, which is a symptom of the acute stage, may be extreme, and continue into convalescence, or last for weeks. I know of one case in which the sweating was profuse and persisted for months, and caused the greatest distress by requiring frequent complete change of clothes both day and night. This case was most refractory. All drugs failed to control it until a happy thought suggested nitrite of amyl, which, strange to say, acted like a charm. In most cases the sweatings stand in direct relation to the debility, and yield to strychnine, general tonics, and change of air.

Treatment.—There is no antidote to the disease, and treatment must therefore be symptomatic. Depression being so marked a feature, the remedies should be in general stimulating and not depressant. The most important fact to be kept ever present in the mind is that care, during the post-febrile stage of depression, may ward off many of the grave complications, upon which the mortality of influenza chiefly depends.

THE RESPIRATORY COMPLICATIONS OF INFLUENZA.

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The chief stress of the attack of the influenza bacillus falls in a preponderating proportion of cases on the respiratory tract. We find that in some of the earliest records of the epidemics of the disease, those of the 12th century, special reference is made to cough as a chief symptom which in these is variously described as *tussis magna*, *tussis intolerabilis et inaudita*, or *tussis gravissima*. We are told that a violent cough disturbed the patients night and day, and the name *coqueluche*, now applied to whooping-cough, was given to the malady by French physicians on this account. Thomas Short, in the 16th century, referred to the symptoms as mainly consisting of most severe cough, pain in the side, difficulty of breathing, and a fever. In some, it turned, he said, to a pleurisy or peripneumony.

The cough, in somewhat later accounts, was described as troublesome with copious expectoration, or as tormenting, or distressing. The cough, it was pointed out, was often accompanied with dyspnœa, sometimes severe. Phthisis, pneumonia, and pleuritis were mentioned as complications.

What has been observed in the past with regard to cough and pulmonary complications has been equally true of the epidemics of the end of last and the beginning of this century. We have observed the same distressing, hard, frequent, short cough, as one of the most common symptoms of the disease, and we have only too often seen the serious complications of bronchitis, broncho-pneumonia, pneumonia, pleurisy, coming on during the height of the disease, or while it ought to be subsiding. We have seen a large proportion of cases, in which tuberculosis has ensued later on.

It is difficult to separate the pulmonary symptoms, which are an actual part of the disease, from those which more properly may be described as the result of complications. The two merge so completely into one another that it is hardly practical to differentiate them, and it would serve no useful purpose to make the attempt, even if one could successfully accomplish it.

We shall first describe the morbid changes which are met with in the respiratory tract, whether as part of the disease or as complications.

The larynx is frequently affected, but very seldom to any great degree of intensity. The vocal cords are usually congested, and sometimes the redness is extreme. In rarer cases, there is swelling, especially of the ventricular band, the cords and sub-glottic tissue or of the arytenoid folds.

The mucous membrane of the trachea is peculiarly liable to be affected. There is diffuse redness and a coating of thick viscid mucus. Pfeiffer's bacillus is present, sometimes in great abundance, and in almost pure culture in the secretion.

The mucous membrane of the bronchi when inflamed shows similar changes. The ciliated epithelium is partly destroyed, and detached fragments are shed into the interior of the bronchi. Some of the cells are separated from their base by leucocytes. The latter line the free surface, lying in small groups on apparently intact epithelial cells, as well as penetrating between the cells, or filling the spaces left by the cells which have been shed. Bacilli are present in great force lying over the epithelial cells as well as between them. The epithelial cells themselves, and the leucocytes which make their way between them, are filled with clumps of bacilli, but in the submucous tissue the bacilli may be met with singly. The irritation, set up by the presence of the bacilli in and between the epithelial cells, leads to hyperæmia of the submucous tissue. The capillaries and venules in the bronchial wall are distended with blood, and the peribronchial tissue is infiltrated with migratory cells. The affection of the trachea and bronchi has been compared with, and appears to be analogous to, that of the urethra and conjunctiva set up by the gonococcus.

In the case of the smaller bronchi, it may be seen that the lumen is more or less filled with finely granular material, consisting of coagulated mucin, in which are embedded numbers of leucocytes. When the alveoli themselves are affected, they contain not only leucocytes, but also numerous swollen cells which are often pigmented. The latter are regarded by Pfeiffer as altered epithelial cells.

Broncho-pneumonia is the most common pathological change in the lungs. It is characteristic of influenzal pneumonia that it occurs in numerous scattered areas of lobular consolidation, which, although affecting an extensive tract, are separated by small air-containing patches. The cut surface has a somewhat mottled appearance, and is usually smooth or only slightly granular. The consolidation is due rather to a cellular inflammation than to a fibrinous exudation, as in ordinary pneumonia. The solid areas more resemble spleen substance than liver substance, and the lung has been said to be splenicised rather than hepatised.

Pfeiffer describes the lobular areas of consolidation as consisting of small patches, varying in size from a pin's head to a pea, separated by greyish-yellow colour from surrounding dark red tissue. Yellowish-grey, thick, and very tenacious pus may be seen exuding from the cut sections of the bronchi at the centres of the consolidated areas. In the consolidated islets, Pfeiffer's bacilli are numerous, and are found partly within and partly without the leucocytes and the epithelial cells. But other micro-organisms, pneumococci, streptococci, or staphylococci are not infrequently present.

At a later stage, there is a tendency to the formation of small abscesses. Some of the lobular areas become entirely filled with leucocytes, and these merge into one another, thus forming small centres of suppuration.

But pneumonic consolidation of the lobar type may also be met with. While broncho-pneumonic consolidation may be considered typical and characteristic, lobar consolidation is a not infrequent complication.

Fränkel's diplococcus, or the streptococcus pyogenes may be found present in these cases. The appearances of the consolidated lung resemble those in ordinary pneumonia. The upper lobes, however, seem to be more often affected than in the non-influenzal cases.

Influenzal pneumonia is slow to undergo resolution. It is important to remember that sometimes the affected areas undergo transformation into cicatricial connective tissue, being then replaced by firm greyish-red fibrous masses, sometimes of almost cartilaginous consistence, and the alveoli are occupied by newly-formed fibrous tissue.

Gangrene of the lung sometimes occurs, as the result of secondary infection by bacteria from the mouth. The pleuræ are frequently affected when the lungs are involved. Flakes of fibrous exudation, patchy or diffuse, varying in thickness as well as in size, may be observed on the surface of the lungs. These consist chiefly of leucocytes containing crowds of bacilli. In other cases, there is a sero-purulent exudation, and the effusion has a peculiar cloudy, rather turbid-yellow appearance, so that it has been likened to clay-water or cream of tartar.

Symptoms.—The chief symptom of the laryngeal affection, which, as has been pointed out, is seldom of much intensity, is hoarseness or aphonia. Even in ordinary cases, the voice may often be noticed to be husky and weak. Soreness in the situation of the larynx is not uncommon. The symptoms are more marked in the rarer cases, in which the laryngitis is of a more severe type.

The affection of the trachea is, however, usually a more distinctive feature of the disease. The diffuse injection of the mucous membrane results in a very severe hacking and persistent cough, which comes on at times in fits, occurring throughout night and day, resembling those of whooping-cough, and causes the patient much distress. There is also pain along the course of the trachea. The cough is often, at any rate at first, quite dry, or attended with very scanty glutinous expectoration.

The cough frequently persists after the acute stage has long passed away It may be so severe and paroxysmal as to lead to rupture of some of the fibres of the abdominal muscles, as in the case of a lady whom I saw in consultation recently. During a fit of coughing, she was seized with pain in the abdomen, and soon after it was observed she had a tumour as large as a cricket ball in the left hypochondrium. The presence of a hard roundish tumour gave rise to a suspicion of carcinoma. I came to the conclusion, however, that the tumour was a hæmatoma in the abdominal wall, and this the course of events proved to be the case, and it gradually disappeared in the course of two or three months.

The bronchi are affected in a large number of cases, and partially so with special frequency. At first, there is merely hyperæmia, so that when the fever has started and the attack is at its outset, there is frequently hardly any sign of secretion. Swelling of the bronchial mucous membrane, however, often follows, and this is manifested by a louder respiratory murmur at certain points, or by a harsher inspiratory sound. The cough is just as severe as when the trachea is affected. The breathing becomes shorter and more rapid, and, at times, there is actual dyspnœa. The presence of scattered patches of atelectasis may give rise to slight dulness on percussion and diminished respiratory murmur. This, the stage of dry catarrh, seldom lasts long. As a rule, it is not long before there is secretion from the mucous membrane, and there is expectoration, which is usually purulent from the first. On listening over the lungs, we find the physical signs of bronchial catarrh with great variety in the character of the râles. The sputum is usually yellow or greenish yellow, and is sometimes tinged with blood, or has a peculiar flesh-coloured tint. It is sometimes nummular, sometimes viscid, and sometimes almost diffluent. It may be profuse, and in amount there may be as much as one or two pints a day. Occasionally, fibrinous casts are coughed up. Occasionally, bronchial dilatation may occur early, and the dilated tubes are filled with muco-purulent secretion.

When the smaller tubes are affected, it is common to hear, at the bases, râles with a characteristic sharp high pitched sound, the "sharp sticky" râles of Goodhart, to which I shall have occasion to refer again.

While tracheitis and bronchitis may be regarded as integral parts of the disease when they are slight, and as complications when severe, the next affection which we have to consider, namely, broncho-pneumonia, must be looked on always as a complication. It is the most frequent pulmonary complication, is always serious, and is always a cause of anxiety. The patient affected by it soon becomes very ill. The pulse and respiration become rapid, and the fever often runs high. Sometimes severe pain in one or other side is complained of, the face is flushed, or congested, or there is actual cyanosis. There is usually much prostration.

At the onset of broncho-pneumonia, there is seldom any initial rigor, but there may be repeated slight chills and sweating later. The pulse in broncho-pneumonia is not only rapid, but it usually becomes very small. There is a greater degree of dyspnœa than one would expect from the rate of respiration, or from the extent of lung involved. Air hunger is often a marked symptom. The temperature follows no definite type, and, as a rule, does not long remain high. It usually is irregular, and varies between 101° and 104° Fahrenheit. In elderly subjects, it is more likely to keep about the lower of these limits, and there is no relation between the severity of the illness and the height of the fever. The fever ends, as a rule, by lysis, not by crisis. Cyanosis sometimes rapidly increases when the illness is approaching a fatal termination. The sputum rarely resembles that characteristic of lobar pneumonia. It is yellowish or yellowish green, and it is tenacious and expectorated with difficulty, but it is never, or hardly ever, rusty.

The physical signs are usually most marked at the bases. There a certain degree of dulness may be made out, and there are fine and coarse bubbling râles, with sometimes distinct but distant bronchial breathing. While, in most cases, the bases are affected, in some the apices are the seat of the complication. In other cases, the pneumonia wanders irregularly, and we may observe, for instance, that first the lower lobe of one lung is affected, and then the upper lobe of the other. The dulness is seldom so pronounced as it is in ordinary lobar pneumonia, unless the broncho-pneumonia has lasted some time. The dull areas are not sharply defined, but gradually merge into the resonant parts. They seldom extend throughout an entire lobe, and specially affect the borders of the lungs. The presence of bronchial breathing is by no means constant. In consequence of the blocking of the bronchi leading to the dull areas, either by swelling of the mucous membrane, or by profuseness of secretion, these breath sounds are often very feeble, or almost inaudible. When the breath sounds are bronchial, they are nearly always distant. The typical tubular breathing, which is heard in lobar pneumonia, is very exceptional.

Special attention has been paid to the râles, which are to be heard over the bases of the lungs, when affected with broncho-pneumonia, already referred to as the "sticky râles" of Goodhart. These râles have been termed by Wintrich vesicular. They give the impression of having originated in the bursting of a large number of air bubbles of a uniform size. They are explained by Wintrich as being produced by the sudden tearing apart during inspiration of the adherent walls of the smaller bronchi and alveoli, stuck together by adhesive mucus. Ordinarily, it is impossible that these walls could stick together, but it is conceivable that it could take place when their mucous membrane is swollen, and is covered with tenacious mucus. This explanation is supported by the fact that even forcible coughing and expectoration have absolutely no effect on the character of these sounds. If this explanation is the correct one, the term sticky râle seems not altogether inappropriate, although sticky is not a very expressive adjective to apply to a sound.

An unfavourable termination of the disease is unfortunately only too frequent. The dyspnœa then increases, the pulse becomes feebler as well as more rapid, delirium sets in, prostration increases, and the patient gradually sinks. Patients who recover mend slowly, and the lung only very gradually clears up. Relapses are not at all uncommon.

While, as a rule, broncho-pneumonia occurs along with and as an extension from bronchitis, it may also be met with without any evidence of bronchitis. There are then the physical signs of consolidated areas in the lung, with abundant fine râles, such as one gets in the early stage of lobar pneumonia, but without any moist sounds or rhonchi. These cases often run their course without expectoration, or there may only be purulent expectoration when resolution is commencing.

While the pneumonia set up by Pfeiffer's bacillus is essentially of the lobular type, we meet with other forms of pneumonia, due to mixed infection. When pneumococcic pneumonia attacks a patient with influenza, it may be observed that the constitutional symptoms are out of proportion to the local lesion. As a rule, pleuritic pains are absent, or are little marked. There is much greater dyspnœa than is generally observed in acute pneumonia. There is a dry periodic cough with sero-mucous, blood-stained expectoration, containing pneumococci. Rusty sputum is often absent. There is a relatively small dull area with weak or absent respiratory sounds, but bronchial breathing and bronchophony are absent, and there are no râles at the beginning. It may be added that an initial rigor is the exception. Profuse sweats are common without a corresponding fall of temperature. The fever runs an irregular course, and terminates more frequently by lysis than by crisis. After the fever has departed there is great weakness left, and the respirations may continue rapid.

Another form of pneumonia which has been observed is what has been called cellular pneumonia. This very often begins quite acutely, invading rapidly an entire lobe, very often an upper lobe. There is no rusty sputum. The cut surface of the lung is smooth and homogeneous, and the consolidated tissue feels hard, elastic, and of a fleshy consistence. There is great hyperæmia. The alveolar septa are thick and infiltrated with round cells, while the alveoli are packed with cells with only traces of fibrin. In streptococcus pneumonia, the inflammation is of this type, being cellular without any of the characters of a fibrinous process. At first, there are many small areas of consolidation which eventually run together to form one.

It has been calculated that pneumonia is met with as a complication of influenza in from 5 to 7 per cent. of the cases. If we were to draw conclusions from our hospital cases, we might think it was a much more frequent occurrence. But it is obvious that only the more severe cases of influenza will be admitted to a general hospital, and that therefore any conclusions as to the actual frequency of pneumonia among a population attacked with influenza would be fallacious. The mortality of influenzal pneumonia appears to be greater than that of ordinary pneumonia. It has been variously estimated at from 17 to 43·9 per cent. of the cases affected.

Pleurisy occurs as a complication in from 12 to 33 per cent. of the cases. As a general rule, it is sero-fibrinous, but it is sometimes purulent. In influenzal empyemas, the streptococcus pyogenes has been found to be present. A form of acute primary pleurisy has been described, which comes on nearly simultaneously with the onset of influenza. This is generally an extremely grave affection, and is characterised by chills, high fever, severe dyspnœa, and cyanosis.

The complications, which have hitherto been described, have been of an acute nature, but chronic affections, chronic bronchitis, and chronic pneumonia may also be set up, and left behind after an attack of influenza. Cough and expectoration persist. At intervals, varying from a few days to a few weeks, catarrhal symptoms wax or wane, without any apparent cause. In these cases, influenzal bacilli continue to be found, over long periods of time, in the expectoration in large numbers and in pure cultures. The general health, in such cases, is extremely variable. Sudden fever may occur, or there may be bouts of perspiration, or there may be attacks resembling asthma.

I have seen several cases, in which the physical signs of consolidation have persisted for several months after an attack of influenzal pneumonia, and then have gradually disappeared.

Pfeiffer's bacillus was found by Netter in the expectoration during a period of fourteen months in a case with induration of lung.

It should also be remembered that influenza may become chronic in patients suffering from pulmonary tuberculosis. Lesions, characterised by fibrosis, with consolidation of large tracts of lung, may result from the chronic form of influenza. In such cases, bronchiectasis may follow later on.

An attack of influenza is given as the starting point in many cases of pulmonary tuberculosis. The patient, debilitated by a severe attack of influenza, has in his lungs a favourable soil for the tubercle bacillus, and offers a lessened resistance to its inroads. In the case of a patient, with a quiescent tuberculous lesion, an attack of influenza appears to excite it to renewed activity.

Treatment.—The respiratory complications of influenza are difficult to treat, and every effort should be made to avert them. In the simplest attack of influenza, the patient should be kept in bed for some days after the fever has subsided. When respiratory complications are present, he should, as a rule, be kept in bed until they have disappeared. The atmosphere of the room should be kept wholesome, and this can only be done by keeping the windows open night and day.

The patient must be kept warm, but he must not be oppressed with too many bed-clothes. The temperature of the room should be not over 62° F.

As we have no specific remedy, treatment has to be based on symptoms, and the first symptom of importance is the cough. A great many remedies have been tried and we cannot say that any remedy is specially efficacious. Where the cough is dry and paroxysmal, heroin hydrochloride in doses of from $\frac{1}{36}$th to $\frac{1}{12}$th of a grain may be given at intervals of from one to two hours. Barley water, linseed tea, toast and water, or warm milk may be sipped occasionally. The following linctus has been found to be efficacious where the cough is frequent and severe :—

℞	Morphinæ Hydrochlor.	gr. ½.
	Apomorphinæ Hydrochlor.	gr. ¾.
	Acid. Hydrochlor. Dil.	m. xx.
	Syr. Prun. Virgin	℥ss.
	Aq. ad	℥ij.

Ft. linct. Sig. ʒj occasionally.

When there is bronchitis, a mixture containing citrate of ammonia, citrate of potash, and ipecacuanha wine is useful.

℞	Liq. Amm. Citrat.	ʒjss.
	Pot. Citrat.	gr. xv.
	Vin. Ipecac.	m. v.
	Aq. ad	℥j.

t.d.s.

Another mixture which is useful is the Mist. Sod. c. Æther. Chlor. of the Brompton Hospital Pharmacopœia. This is :—

℞	Sod. Bicarb.	gr. xv.
	Sod. Chlorid.	gr. v.
	Sp. Chloroform.	m. v.
	Aq. Anisi	℥j.

t.d.s.

When the secretion is thick and viscid, and expectorated with difficulty, preparations containing opium are contraindicated, and should not be given. Linseed or linseed and mustard poultices or turpentine stupes sometimes give much relief, when there is tightness and soreness of the chest.

The same remedies may be given in cases of pneumonia. Here the main indication is to maintain the patient's strength.

The chief danger is cardiac failure, and when there are signs of failing heart, stimulants should be cautiously administered. Digitalis, strophanthus, caffein, and strychnine are all useful drugs.

Huchard recommends 30 to 50 minims per diem of a solution of digitalin (1 in 1000). Finkler advises the use of camphor in heart failure, camphorated oil being given subcutaneously.

Oxygen inhalations will relieve when there is marked dyspnœa.

Alcohol is indicated when there is prostration and failing heart, and champagne and brandy are specially valuable. The amounts to be given must be regulated by the effects on the heart and pulse in individual cases.

The diet should consist mainly of milk, eggs, beef-tea, chicken broth, rusks, arrowroot, Benger's food, and calf's-foot jelly. The hygiene of the mouth and teeth should be particularly attended to. The bowels should be acted on if necessary.

Fresh air is the best prophylactic against influenza. It is also a sovereign remedy in the treatment of both the acute and chronic manifestations of the disease in the respiratory tract. Nothing is more striking than the way in which influenza spreads in the stuffy and ill-ventilated hotels, in which people have usually to live who travel in search of health. Out of many hotels in a well-known health resort, I know of one only in which the visitors escaped influenza during several winters, and that was a hotel in which, thanks to the presence of a doctor, who knew the value of fresh air, the windows in the public rooms and lounges were kept constantly open. How long it will be before the general public in this country will come to recognise that open windows are health-giving, instead of being dangerous, I cannot tell. The people who have been brought up with the idea that the slightest breath of air, which comes into a house from outside, may give them a chill are not likely to change their views for all our assurances that they are mistaken. These are the people who insist on closed windows in trains and hotels, and in all places of public resort, and it is through their mistaken prejudice that influenza spreads as it does.

But this is by the way. What I want to insist on is the advantage which accrues to the influenza patient if he is treated rationally and sensibly, and if the air of the sick room is rendered wholesome by free opening of windows.

Many doctors, I think, still cling to the old ideas that bronchitis, broncho-pneumonia, and pneumonia must be treated in a warm room, and that it is dangerous to allow the temperature of the room to get below 62° F.

But purity of the air, which the patient gets to breathe, is of far greater importance than its temperature. In fact, up to a certain point the cooler air is, the more invigorating and refreshing it is, and it is senseless to deny to the patient fresh air from outside, and allow him a draught of cold oxygen from a cylinder.

Open-air treatment is the best restorative in chronic influenzal respiratory affections, and I am quite as much impressed by the results obtained therefrom in these troubles as in chronic pulmonary tuberculosis.

I would that there were some hotel or sanatorium, run on open-air lines, in one of our own South Coast health resorts, for the reception of non-tuberculous patients convalescing from influenza, or from some other acute illness. It would supply a felt want, and would do much to promote the rapid and complete recovery of its visitors.

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INFLUENZA AS IT AFFECTS THE NOSE AND THROAT.

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THE term influenza has, unfortunately, been applied to various forms of catarrh, so that many have come to regard an "influenzal catarrh" as a necessary first stage of this affection. No doubt the infection of influenza does enter the system through the upper air passages; in this region many of its most typical effects may appear; and a naso-pharyngeal catarrh is a usual but not necessary part of the manifestations of the disease. In the upper air tract the influenzal virus shows itself by (*a*) inflammatory affections or (*b*) neuroses.

INFLAMMATORY AFFECTIONS.

The Nose.—A spontaneous epistaxis may usher in the symptoms of influenza. In the early stage an acute rhinitis may occur, which, except by the presence of general symptoms (rigors, pains, fever), cannot be distinguished from an ordinary acute catarrhal rhinitis. The occurrence of this "influenza cold" varies in different epidemics. In my own experience it is a less frequent occurrence than in the visitation of fifteen years ago. It is more usual to meet with a typical septic infection of one or more accessory sinuses, and it is more likely to occur after a sore throat, or general symptoms have manifested themselves. The patient then states that, after the onset of influenza, a profuse muco-purulent or purulent discharge appears from one or both nostrils, while the neighbourhood of the orbit may become tender and puffy, and severe pain may radiate along the first and second branches of the trigeminal nerve. It is this pain which is so frequently treated as simple "supra-orbital neuralgia" or "face-ache," without its being recognised that the real cause is an inflammatory process in the subjacent frontal, ethmoidal, or maxillary sinuses. Sometimes the symptoms round the orbit appear, although no pus can be discovered in the nose. In one case, an abscess formed over the right frontal sinus in a boy of eleven, who had a "slight cold," was out of sorts, and complained of a sore throat. Three days later there was tense œdema, without redness, of the right eyelids, and tenderness at the upper and inner angle of the right orbit. No pus could ever be discovered in his nose, and yet, on incising the swelling, there was an escape of free fluid pus, welling out from the right frontal sinus, and containing the *Micrococcus catarrhalis* in pure culture.[1] The eyelid may swell up and close in a single night.

Severe pain, referred to the neighbourhood of the orbit in these cases, may not disappear entirely with the cessation of the discharge. A woman, aged 30, had influenza last April with severe pain on the left side of the forehead. She reported that the left eye gradually closed up a month before admission to the hospital, but opened of itself after a week. She had suffered from a bad tasting discharge from the nose, requiring the use of as many as 12 handkerchiefs a day. When seen two months after infection there was no anosmia or cacosmia. No pus could be discovered in the nostrils, though a small trace was occasionally seen in the naso-pharynx ; both maxillary sinuses were quite clear on transillumination. Yet this patient presented puffiness of the left eyelids, and continued to complain of pain in the region of the left frontal sinus, with extreme tenderness on pressure. As the symptoms were not urgent, I waited a fortnight before removing the anterior end of the left middle turbinal. This gave no relief, and a few days later I opened the left frontal sinus. It was perfectly healthy, and, as transillumination had excluded the maxillary sinus, it was clear that her symptoms were due to some retention in the ethmoidal cells. She continued to manifest swelling, tenderness, and redness of the left upper eyelid, while the inner angle of the supra-orbital arch remained very tender, so that, although we kept her in hospital for another four weeks, she was sent to the country without being relieved. A fortnight later she reported herself better, and when seen three months afterwards all pain and tenderness had disappeared.

If the sphenoidal sinus is affected, the symptoms will consist chiefly of post-nasal discharge, pain referred to the occiput, or deep between the eyes, and, not infrequently, marked somnolence. The sinus infections from influenza are generally relieved as soon as there is a free discharge of pus from the nose. But, in many cases, both the acute and the convalescent stage may occupy some time. Fig. 1 shows

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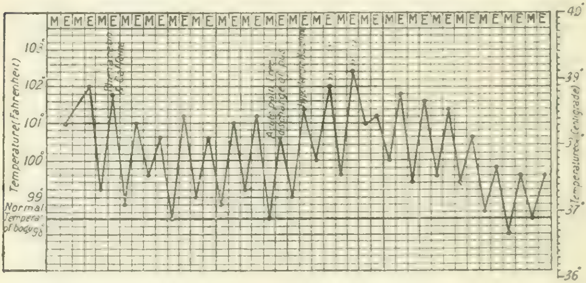
[1] *Vide* THE PRACTITIONER, July, 1906.



the temperature chart of a nurse, who was in my ward for three weeks, with an acute influenzal sinusitis of the cavities of the left side. At the end of six weeks she was discharged convalescent, but with neuralgia and tenderness of the left frontal sinus, obscurity to transillumination of the cavities of the same side, and pus in the left nostril. It required another six weeks of change of air before all these symptoms completely and permanently disappeared. Another nurse was warded at the same time, with infection of the accessory sinuses on the left side, and, a few days later, acute suppurating otitis media on the same side. In her case, the sinusitis entirely cleared up at the end of a month, but, after a month of change and local measures to the ear, my colleague, Mr. A. Cheatle, found it necessary to open her mastoid. Her illness from beginning to end cost her exactly four months. I have seen her lately; all her accessory cavities, including the middle ear, are quite normal.

In a case, seen in consultation with Dr. Ernest Snape, there was a unilateral sinusitis, and an acute otitis media. The drum was incised and healed up completely in three weeks. The nasal suppuration, on the other hand, dragged on for some four months before it entirely disappeared. The above cases were evidently primary influenza infections of the nasal accessory sinuses. But we must be prepared for cases, in which the patient may have a latent empyema, which the fresh infection lights up temporarily. In a man, seen at the Seamen's Hospital, the right nostril was full of pus and polypi, and on removing the latter, the condition of the orbit returned to normal. We then found a chronic suppuration of the frontal, ethmoidal, and maxillary cavities, evidently of old standing.

Pharynx.—A diffuse inflammatory condition of the naso-pharynx and pharynx frequently occurs in influenza. Lacunar tonsillitis is not uncommon, and the deposit in the lacunæ is apt to run together and to form a false membrane extending even on to the fauces. Quinsies may also form.

Larynx.—Acute catarrhal inflammation is very common in influenza. Painful hoarseness may be due to inflamed, and even abraded cords. Or the vocal cords may not be affected so that the patient's voice is but slightly altered, while he complains of the raw or burning pain, a swollen feeling, and difficulty in swallowing. This is due to the fact that the other parts of the larynx (ventricular bands, ary-epiglottic folds, and particularly the arytenoids and epiglottis) are found to be swollen and deeply congested. In rare instances, acute septic œdematous laryngitis occurs which may lead to extensive ulceration, sloughing, and necrosis; and other forms of septic sore throat have been met with. This is an extremely fatal complication.

NEUROSES.

Nose.—Anosmia is so apt to be a sequel of influenza that, when it occurs associated with any suspicious symptoms, I look upon it as one of the chief diagnostic features. In the absence of pathological changes in the nose, recovery will generally take place, even after many months. But in some cases it is permanent. Cacosmia will generally be found not to be a neurosis, but due to suppuration in one of the accessory sinuses. This is most frequent when the maxillary cavity is affected, and when there is also infection from the teeth.

Pharynx.—Paralysis of the soft palate has been observed, and some dysphagia may be due to weakness of the constrictors of the pharynx. A condition of hyperæsthesia or anæsthesia is not unusual.

Larynx.—Paresis of the tensors of the vocal cords may remain after an influenza laryngitis. Paralysis of one or both vocal cords is not uncommon ; as usual, it is the abductor form which is met with. If only one cord is affected, the voice is only slightly altered. This paralysis is doubtless due to peripheral neuritis, and, as there is nothing distinctive about it, the diagnosis has to be carefully made from the numerous other possible causes of a lesion of the recurrent laryngeal nerve. Sometimes the paralysis will clear up in a rapid and surprising way ; but, in other cases, I have watched it remain permanently for years.

Cough.—A barking, inveterate cough is another legacy of influenza. Sometimes it consists of a long drawn whoop, followed by a succession of exhausting, explosive, expiratory coughs. This is frequently referred by the patient to the region of the trachea, and, while it is possible that the point of irritation may be situated in the wind-pipe, we have no means of settling whether it may not be in the larynx or pharynx. Not uncommonly, these coughs, left after influenza, owe their origin to pus in the accessory cavities trickling backwards into the naso-pharynx. This auto-intoxication may induce many symptoms suggestive of phthisis :—loss of flesh, sweats, rise of temperature, dyspepsia, expectoration of pus, etc. Even alteration of the pulmonary breath sounds may occur, with diminished expansion. In such cases the examination of the sputum, and the tracing of pus to its true origin, determines an exact diagnosis.

Bacteriology.—I do not know whether pathologists would regard it as very unorthodox, but I venture to suggest that the clinical symptoms of influenza in the upper air passages may be brought about by more than one variety of micro-organism. In the case of the boy, mentioned above, the pus yielded the *Micrococcus catarrhalis* in pure culture. The two nurses referred to developed influenza at the same time, and were warded alongside of one another. One, in whom the sinusitis took three months to clear up, showed *pneumococci* in pure culture ; while, in the other, in whom the sinusitis subsided within a month, but whose ear infection required a mastoid operation, the pus from the nose yielded the *influenza bacillus* in pure culture. These observation were controlled by Dr. Emery. Space does not permit of going into this question at present, but it seems to deserve consideration.

Treatment.—The general treatment is of primary importance in the inflammatory infections. I have little doubt that many cases of acute sinusitis drift into chronic empyemata through neglect; while all the cases which I have seen treated from the commencement have cleared up. The patient should remain in bed until the acute stage is past; he can be kept very warm, but with his bedroom windows widely open. Tenderness or neuralgia is met by having the head wrapped up in a woollen shawl; hot fomentations are applied over the affected side, or the patient may lie with his face on a rubber hot-water bottle. If the maxillary sinus is affected, and there are any suspicious bicuspid or molar teeth on the same side, they should be removed. The pain can be met with phenacetin, caffein, aspirin, or similar drugs, but when severe, there need be no hesitation in giving hypodermic injections of morphia. As regards local measures, it is well to carefully avoid the use of sprays or lotions during the acute stage. They are apt to disseminate the infection, and there is the risk of their carrying it to the ear. It is better to prescribe the following :—

℞.	Menthol. - - - -	½ dr.
	Tincture of Eucalyptus - -	3 oz.

A teaspoonful of this is put in a pint jug of steaming water, and the vapour is inhaled up and down the nose every two or three hours. It generally relieves pain, it frequently stimulates a free discharge, and patients are satisfied that it "clears the head." In view of cases, in which I have tried it, and the results published by Logan Turner,[1] I think relief can safely be obtained by puncturing the suppurating antrum from the nose, and washing it out with a tepid sterile saline solution. Relief to the frontal and sphenoidal sinuses may be secured by packing the neighbourhood of the middle meatus, for a few minutes every day, with a pledget soaked in cocaine (5 per cent.) and adrenalin. Experience shows that these sinus suppurations are more apt to occur on the side of the nose which is the more obstructed (by deviations, spurs, etc.); if the middle turbinal is very crowded, and evidently acting as an obstruction to the vent of discharge, its anterior portion can be removed. When the acute stage is past, a simple alkaline nose lotion may be employed. As the above cases have shown, there is no need to despair of ultimate resolution till change of air, rest, hygiene, and tonic treatment have raised the resistance of the patient. The inflammatory troubles of the pharynx and larynx are treated on ordinary principles.

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[1] *Edinburgh Medical Journal*, August 1906, p. 152

Neuroses.—A guarded prognosis should be given as to the relief of anosmia. Beyond seeing that the nasal cavities are in a healthy condition, it is well to refrain from local measures. All the nasal alkaline lotions containing carbolic or the usual volatile antiseptics, like menthol, camphor, eucalyptus, thymol, etc., should be avoided. A long-continued course of strychnine, together with general measures, is more likely to be successful. The same line of treatment applies to neuroses of the larynx, but paralytic conditions of the cords may be improved by the use of electricity. On the whole I have greater faith in general treatment.

The paroxysmal cough is sometimes obstinate. Sedative sprays and lozenges of menthol, heroin, codeia, or morphia, should be tried. Cocaine is best avoided, as the anæsthesia produced is alarming and disagreeable to the patient. These cases sometimes include what we regard as "stomach coughs," and doubtless there is a change in some of the other branches of the pneumogastric. Morphia and opium, given by the stomach, are often, therefore, extremely useful; the effect of bismuth may be tried; and Warburton Begbie's mixture can be recommended:—

℞	Diluted Hydrocyanic Acid - -	½ dr.
	Diluted Nitric Acid - - -	½ dr.
	Glycerine - - - - - - -	½ oz.
	Infusion of Quassia - - -	6 ozs.

A tablespoonful in a wine-glass of water three times a day.

While the various forms of linctus and syrup are helpful, it will generally be found that some suitable mixture will be effective, while, for those who can afford it, there is nothing better than rest and change.

INFLUENZA IN RELATION TO THE DIGESTIVE ORGANS.

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THE ALIMENTARY CANAL.

It is necessary, in the first place, to point out that it is extremely difficult to prove that any particular case of gastro-intestinal disturbance is due to influenza. Even when we are dealing with affections of the nasal or bronchial mucous membranes, such proof may not be easy unless the other symptoms, the sudden onset, the great mental and physical depression, the pains in the limbs, etc., are clearly marked, or unless we find Pfeiffer's bacillus in the discharges. But, in affections of the alimentary canal, the difficulty is almost insuperable, for the characteristic general symptoms are often absent; indeed, there is reason to believe that an influenzal gastro-enteric catarrh may exist without any rise of temperature. Then as regards Pfeiffer's bacillus, the chances of finding it in the discharges from the stomach or bowels are infinitesimal, and I can find no case in which a search for it in the stools has been successful. As an instance of the difficulty, it may be mentioned that, in the early days of the 1889–1890 epidemic, when search was being everywhere made for the specific organism, Weichselbaum, in describing the post-mortem on a case of acute enteritis, which was supposed to be due to influenza, states that the bacteria in the ileum (the most affected part) were so numerous that he could not isolate any special variety. Again, when we endeavour to detect the cause of a gastro-intestinal affection, we find that the possible sources of error are very numerous. In this connection, I would cite two extracts from the *British Medical Journal.* On January 4th, 1890, this journal, in a note on the epidemic of influenza, which was then prevailing on the Continent, states that such cases as had, so far, been met with in London, were chiefly of the gastro-intestinal type; and again, on August 30th of that year, while discussing the epidemic in Vienna, it states that the cases were chiefly gastro-intestinal. Now in both of these instances, it is doubtful whether the cause of the diseases was really influenza, for the week before January 4th is a period when the effects of Christmas festivities are likely to be evident, and, at the end of August, in Vienna, the usual causes of summer diarrhœa were probably rife. It follows, from what I have said, that it cannot be absolutely proved that all the cases mentioned in this paper were really due to influenza. They have been, however, attributed to influenza for one or other of the following reasons: (1) they occurred during an epidemic of influenza; (2) they could not be traced to any other cause; (3) they presented certain peculiarities in their course, or symptoms, which suggested influenza; (4) their features were altogether so anomalous that the cause could only be some poison, such as that of influenza, which is notorious for producing eccentric effects, particularly upon the nervous system. The only perfectly satisfactory cases would be those which were met with during an epidemic in a large institution, for here it would be practically certain that there was only one poison at work, producing in one person a bronchial, and in another, a gastro-intestinal affection.

On the whole, it may be said that the gastro-intestinal affections of influenza are relatively few. In the published accounts of the various epidemics, they are often passed over in silence, or it is merely stated that "other cases were of a gastro-intestinal type." In the carefully watched epidemic at King Edward's School for Girls, a report of which appeared in the *British Medical Journal* (1890, I., p. 418), Dr. Hubert Bristowe states that 21 out of 175 cases (12 per cent.) had diarrhœa and abdominal pain, and Kehl (*Deutsch. Med. Woch.*, 1890) found that diarrhœa occurred in 12·9 per cent. of his cases. Dr. Althaus, in his well-known book, estimates that gastro-intestinal cases of all kinds furnish about 15 per cent. of all cases of influenza. Some epidemics, however, show more of these cases than others. It may also be said, though with some reservation, that, on the whole, the gastro-intestinal affections of influenza are not very fatal. It is true that some cases, in which vomiting and diarrhœa rapidly develop, exhibit most alarming symptoms of cardiac depression, and that in those prolonged cases, which simulate typhoid fever, the toxæmia may produce a condition of the greatest gravity.

Yet the total mortality does not seem to be great. Dr. Peacock, who is quoted in Dr. Symes Thompson's book, states that, in the epidemic of influenza of 1847 and 1848, diarrhœa and dysentery were less fatal than at other times, a statement which, by the way, seems to confirm the idea that they were really due to Pfeiffer's bacillus, and not to that of Shiga or Flexner, for a difference in the fatality suggests a difference in the cause. Again, Dr. Reiss (*Die Influenza Epidemic*, 1889 *and* 1890, by Leyden and Guttmann) states that during the epidemic slighter disturbances of the alimentary canal were common, but more severe ones rare. Further, during an epidemic of influenza, the total mortality from gastro-intestinal disorders does not increase, whereas, as is well known, that from diseases of the respiratory organs runs up very considerably. Dr. Dixie, in his report on the epidemics of 1889, 1890, and 1891, published in 1892, draws attention to this circumstance, and, in the *British Medical Journal* (1898, I., p. 513), there is a note to the effect that "though this epidemic is largely gastro-intestinal, there is no increase in the death-rate from diarrhœa or other intestinal diseases, in fact it is even below the average." Finally, Frohlich (*Wien. Med. Wochens.* 1892, Vol. 42, p. 302) says that the prognosis is better in influenza than in the gastro-intestinal affections which it simulates. Now, if the gastro-intestinal affections caused by influenza are not very numerous, and not very fatal, it might be thought that a consideration of them was not very important. But this is not the case, for it is an undoubted fact that, during an epidemic of influenza, anomalous cases of gastro-intestinal disease occur, which closely simulate cholera, typhoid fever, and, to a less extent, dysentery. The importance of knowing this fact and of attempting (however difficult the task may be) to diagnose the influenza from the other affections at an early period of the disease is obvious, if it were only from the point of view that the prognosis in influenza is more favourable. But there is a side to this question, which is even more serious, for there are cases of influenza which simulate such acute affections as gastric-ulcer with perforation, appendicitis, and intestinal obstruction, and, as the symptoms present might induce a surgeon to operate, and as unnecessary abdominal section during influenza would greatly prejudice any hope of recovery, the importance of the subject can scarcely be over-estimated.

We will consider (1) the part, which symptoms, referable to the alimentary canal, play, in the course, of what we may call ordinary cases of influenza, and (2) those cases in which the influenzal poison acts chiefly upon the alimentary canal and the nerves which supply it.

I.—THE SYMPTOMS, REFERABLE TO THE ALIMENTARY CANAL, WHICH OCCUR IN ORDINARY CASES OF INFLUENZA.

The "tongue" does not present any characteristic appearance. It tends to be furred and dry as in most cases of fever. In severe, prolonged cases with high fever, it becomes brown and tremulous, as in the typhoid state. It has been stated that, if abdominal symptoms are present, its covering is peculiarly thick and creamy. "Anorexia" is present as in all fevers. In some cases, the appetite returns on defervescence, but, in others, it remains absent for a considerable time. "Loss of taste" is often a marked symptom and sequela of influenza, and it appears to be due to a special action of the poison on the nerves of taste, for it occurs in cases, which have had no prominent gastric symptoms, and, even if such have been present, it may persist long after they have disappeared. I have recently seen a patient, who asserts that his sense of taste has been lost since an attack of influenza six months ago. That the loss of taste is independent of any stomach affection is further shown by the fact, so well known to many of us, that after influenza tobacco is as tasteless as food.

"Nausea" and "vomiting" sometimes occur as initial symptoms. From a consideration of the reports of various localised epidemics, vomiting seems to take place in about 20 per cent. of all cases. Should it persist, some special affection of the stomach should be suspected. In connection with symptoms of onset, it should be noted that "hæmatemesis" and "hæmorrhage from the bowels" may occur without the affection becoming, subsequently, a specially abdominal one. Epistaxis, however, appears to be the most common hæmorrhage of onset. In the epidemic at Funfkirrchen in

Hungary, in November, 1890, many cases had epistaxis and intestinal hæmorrhage (*B.M.J.*, 1890, II., p. 1256). "Flatulence" and "colic" have been frequently observed, particularly by Professor Révillard during the epidemic at Geneva in 1890, and these symptoms are of interest, seeing the frequent association of dilatation of the stomach with pneumonia, and the peculiar attraction of the poison of influenza to the vagus and sympathetic nerves. In ordinary cases "constipation" is the rule, and it may be persistent, or it may give way to diarrhœa. Drs. Robertson and Elkin, in their report on the epidemic at the Royal Asylum, Morningside, Edinburgh (*B.M.J.*, 1890, I., p. 228), state that diarrhœa was frequently started by the administration of laxatives. With regard to the "diarrhœa," it has sometimes (in ordinary cases) the old-fashioned characteristic of being apparently eliminatory, *i.e.*, its appearance has been followed by the disappearance of the other symptoms. This has been noted by several observers, including Dr. Peacock, in his account of the epidemic of 1847 and 1848 (*op. cit.*), and by Dr. Thresh, in his report on the outbreak of abdominal influenza at Ford End in Essex (*B.M.J.*, 1893, I., p. 424). This peculiarity was well marked in a patient, who was under my care, in January, 1892. This gentleman had ordinary influenza with moderate fever, and abundant, somewhat consonating crepitation at the base of each lung. The fever passed off in eight days, but the physical signs in the lungs persisted for 17 days more in spite of all treatment. Suddenly, without the administration of an aperient, he had a sharp attack of diarrhœa, after which the physical signs cleared up with the greatest rapidity.

II.—CASES IN WHICH THE INFLUENZAL POISON ACTS CHIEFLY UPON THE ALIMENTARY CANAL AND ITS NERVE SUPPLY.

Before describing these cases, I would like to draw attention to what seems to me to be a peculiarity which is often present, namely, that the affection begins in the stomach, and then, some days later, passes on to the intestines. We find vomiting and constipation for two or three days, and then diarrhœa. This may be observed in both slight and grave cases, perhaps more markedly in the latter; and it suggests that the poison works its way through from the stomach to the rectum, like an ordinary irritant, such as improper food. But, in the case of common irritants, it is most unusual for a period of several days to elapse before the intestines show signs of irritation ; in fact, the diarrhœa usually sets in early. Hence, if the influenza bacilli act like common irritants, *i.e.*, if they infest and irritate the mucous membrane of the alimentary canal locally, we must believe that they are retained in the stomach for a period of several days, or that fresh growths of bacilli develop periodically (after the manner of the malarial parasite), the original stock attacking the stomach, and a second stock (which develops only after some days) the intestines. Both these hypotheses appear unlikely, and it seems more probable that the bacilli are in the blood, and that their toxins affect the stomach and intestines successively, probably by successively attacking the nerves which supply them, working from above downwards, the pneumogastrics first, and then the sympathetics. That the influenzal toxin does act in this sequential manner is proved by the often-quoted case (Dr. Strange, *B.M.J.*, 1890, II., p. 029), in which the different branches of the vagus were affected on successive nights. The patient suffered on the first night from serious cardiac embarrassment (pulse 20, feeble, and irregular, cardiac oppression and sense of impending death), on the second night, from paroxysmal cough, and, on the third night, from vomiting, which lasted several days. This train of events is held to indicate that the cardiac, pulmonary, and gastric branches of the vagus were in turn poisoned. A case, to which I shall draw attention at the end of this paper, also strongly suggests that the nerves supplying the stomach and intestines were successively poisoned. The idea that in certain cases of influenza, the poison does not go through the alimentary canal as a local irritant, but acts through the blood, is supported by the reports of certain post-mortems on cases which had died with severe intestinal symptoms, and in which the mucous membrane had the appearances seen in general toxæmia (petechial hæmorrhages, etc.) rather than those of ordinary enteritis. Whether the toxin acts through the nerves or not, I think that it can be maintained that a distinct demarcation in time between the gastric and the intestinal symptoms is very characteristic of influenza; and that, in this respect, influenza differs from an ordinary gastro-intestinal irritant, which passes uniformly through the alimentary canal, and also from the bacillus of typhoid fever, which passes through the stomach and upper part of the small intestine without producing any effects therein. The case of the German soldier mentioned in Althaus' book (p. 207) illustrates the above points very well. He was one of a series of 19 cases of enteritis, and was taken suddenly ill with fever and pains in the limbs. The next day there were purpuric spots on the limbs, and, a few days later, abdominal pain and vomiting, the vomit containing fæcal matter and decomposed blood. This lasted for eight days, during which there was absolute constipation. Then suddenly diarrhœa set in, the stools containing at first tarry and afterwards fresh blood. Here there is a distinct demarcation in time between the gastric and the intestinal symptoms. The patient was carried off by pneumonia on the 10th day, and at the post-mortem, the mucous membrane of the intestines was thickened and gray, except for the presence of large numbers of sub-epithelial petechiæ. Petechiæ were also found beneath the skin and in the serous membranes, showing the general toxæmia. There were no intestinal ulcers.

I will mention here another eccentricity, somewhat of the same kind, with which influenza has been charged, namely, that not infrequently there is a relapse in which the symptoms are even more grave than in the first attack. This is a familiar occurrence in ordinary cases. A patient has been ill with fever, bone pains, etc., and, after a few days has appeared to be well. Then a relapse, accompanied by definite pneumonia, has set in. The same course of events may be met with in abdominal cases. Dr. Peacock (*op. cit.*) speaks of this tendency to relapse, and says that the recurrence of the fever may be accompanied by severe diarrhœa with blood in the stools; and in Frohlich's cases (*v.i.*) the relapses were well marked.

The cases, in which the influenzal poison acts specially on the alimentary canal, may be classified as follows:—

(1) Catarrh of the stomach, or of the intestines, or more frequently of both in the same attack.

(2) Inflammation of a more severe type, affecting the same parts.

(3) Cases which simulate typhoid fever.
(4) Cases of a choleraic type.
(5) Cases which simulate ulcerative colitis.
(6) Cases of anomalous type.

(1) *Gastro-Intestinal Catarrh.*—If, during an epidemic of influenza, a patient is suddenly attacked with fever and vomiting, and if no common cause for the symptoms can be found, it may be fairly assumed that he is suffering from influenzal catarrh of the stomach. The diagnosis would be confirmed, if he had headache and severe pains in his back and legs, and if his mental and physical depression were out of proportion to his actual symptoms. In a typical case, the vomiting would persist for two or three days, and it would be accompanied by constipation. In some cases, recovery would then ensue, in others, diarrhœa would set in, perhaps after the administration of an aperient, and, in its turn, would last two or three days. If the gastric and intestinal phases were distinctly demarcated in point of time, the case would be more characteristically influenzal. There would be nothing characteristic about the vomit or stools, both would contain mucus of fluid consistence, that from the bowel being the thinner of the two. Enlargement of the spleen and slight jaundice might be observed. The fever would be moderate and show slight remissions, the exacerbations not being necessarily nocturnal. The average duration of such a case would be from three to eight days, and it would be followed by exceptional debility, and perhaps loss of taste. In some cases the stomach symptoms might be trivial, and the intestinal ones more marked.

(2) It is necessary, on pathological grounds, to make a distinction between the simple catarrhal cases and those of severe enteritis, but, at the bedside, the line of demarcation cannot be strictly drawn. In fact, clinically, cases of enteritis may be divided into those which present the symptoms of catarrh in a very severe form and which last a few days, and those which last longer, and by developing toxic symptoms, give a resemblance to typhoid fever. In the latter, it is impossible, without a post-mortem, to say whether the lesion is actually enteritis; for in some of the fatal cases, as I said before, the bowel presents the appearances seen in general

toxæmia. Putting aside the typhoid-like cases for the present, we may say that, in cases of enteritis, the fever and pains, the vomiting and diarrhœa, are more marked than in the catarrhal form, and that there may be a great deal of abdominal pain and sometimes tenesmus (Ord.). The abdomen is usually distended, occasionally the vomit and stools contain blood, probably from capillary hæmorrhages rather than from ulceration. In the case (*B.M.J.*, 1890, I., p. 322) in which Weichselbaum found after death a typical enteritis, the symptoms were fever, abdominal distension, ileo-cæcal pain and diarrhœa, but pulmonary and cerebral symptoms and lesions were also present. Korman, quoted by Frohlich (*op. cit.*), gives a good idea of what is usually seen after death in the cases that are more severe than a simple catarrh. He says that, in addition to the catarrh of the mucous membrane of the stomach, the viscus shows bright injection in spots, and in a network, and also ecchymoses, and the intestines present the same appearances with, in addition, some swelling of the solitary glands and some flat scabs (presumably coagulation of the upper part of the mucous membrane).

(3) *Cases which simulate Typhoid Fever.*—These have been noted by many observers. The similarity consists in the long duration of the fever, in the presence of abdominal symptoms, especially of diarrhœa with, perhaps, blood in the stools, in enlargement of the spleen, and, lastly, in the development of the symptoms of the typhoid state. G. Monié (*Bull. Med.*, Paris, 1901, XV., p. 154) records a case in which the rose spots were observed. One of the cases reported by Frohlich (*Wien. Med. Wochens.*, 1892, 42, p. 302) is of great interest, as it illustrates the tendency of influenza to a relapse, in which the symptoms are graver than in the original attack. A young woman was admitted to the wards on December 24. She had been ill for one week with fever, debility, metrorrhagia, and constipation, for which she had vainly taken aperients. Her temperature varied from 100° to 102°, and slight diarrhœa was set up by a dose of Ol. Ric. On January 4, she had had no fever for three days, and insisted on going home, though she still had slight diarrhœa. On the day of her return home, she had a rigor, followed by prostration and constipation. She returned to the hospital on January 7, and was found to have a tempera-

ture of 104°. She then became extremely ill with semi-coma and delirium: and diarrhœa set in, the stools sometimes containing blood. She remained very ill, but eventually showed signs of improvement. The diarrhœa stopped on January 17, and, on the morning of January 19, the temperature was for the first time normal. Here we have a five weeks' fever divided into two stages, with delirium, brown tongue, etc., and with diarrhœa and hæmorrhage; altogether bearing a close resemblance to typhoid fever. Frohlich considers that the first attack was one of ordinary influenza, and that the second, which was ushered in by a rigor, was a specially abdominal form of extraordinary severity, and he believes that she had been infected by her brother, who had had a similar illness (including the relapse) away from home, but had returned to his home before he was well.

It is not easy to lay down rules for the differential diagnosis of these cases from typhoid fever. The sudden onset in influenza is important, particularly the sudden onset of the relapse; for although, of late, I have seen many patients with typhoid, who describe the illness as having a sudden onset, I have never met with a true (not an intercurrent) relapse in typhoid, in which the fever did not come on gradually, and in the classical manner. A strong Widal's reaction would be conclusive, but it may be delayed until the 14th or 21st day, particularly in cases which appear to have an intercurrent relapse. The regularly remittent temperature course, as seen on the chart of a typical typhoid case, might be of use, as in influenza great irregularities are observed. Finally, rose spots are very rarely seen in influenza, the stools are not always yellow, the duration seldom extends beyond 14 days, and in some cases defervescence is by crisis.

(4) *Cases of a Choleraic Type.*—It may be considered to be well established that influenza may produce an illness with violent watery discharges from the stomach and bowels, which set in with great suddenness, and produce the greatest prostration and even collapse. A case of this kind is mentioned in Allbutt's *System of Medicine.* In the epidemic of 1893, groups of choleraic cases were met with in Essex, both in the winter and in the summer. If no fault can be found with the food supply, and if Koch's comma bacillus and Gaertner's bacillus

enteritidis are absent, it would not be unfair to attribute such cases, if epidemic, to influenza.

(5) *Cases which simulate Ulcerative Colitis.* It is difficult to speak with certainty as to whether the influenzal poison, ever really produces this affection, because there still exists a loose practice of speaking of any febrile case (except typhoid) in which hæmorrhage from the bowel occurs as one of dysentery, and the post-mortems, which I have quoted, show that, in influenza, blood may be found in the stools without there being any ulcer in the intestines. However, Dr. Peacock (Symes Thompson, *op. cit.*) states that dysentery was met with in 1847–48; and Dr. Frank Nicholson (*B.M.J.*, 1891, I., 1274) records cases with diarrhœa, accompanied sometimes by the discharge from the bowel of mucus, membranous flakes, blood, and sloughs. It may be noted that the reports of epidemics in lunatic asylums (places where ulcerative colitis is prone to occur) do not show any special tendency on the part of influenza to attack the colon.

(6) *Anomalous Cases.*—These are too numerous to speak of at length. There is a case (Dr. Handford, *B.M.J.*, 1891, I., p. 1035) which simulated intestinal obstruction. The patient, a lady of 74, was suddenly taken with abdominal pain and vomiting. The vomit soon became very foul. Next day there was slight fever and the pulse was feeble and irregular, but, after an enema, she rapidly recovered. In another case (*B.M.J.*, 1891, I., 1275) the sudden onset of vomiting with fever and some collapse, and the prominence of pain and tenderness in the right iliac fossa, suggested that the case was one of appendicitis, and in another (*ibid.*) the same symptoms, including the collapse, but with epigastric instead of right iliac tenderness and distension, suggested gastric ulcer with perforation. One thing is particularly worthy of note, namely, that, while all gastro-intestinal affections produce cardiac depression, those due to influenza have this effect to an extreme degree. Thus we have cases of fainting in the watercloset reported, and cases of extraordinary slowness, or feebleness, or irregularity of the heart's action.

Finally, I wish to put on record the following remarkable case. A gentleman, aged about 34, suffered, for a week previous to December 6, 1902, from shiverings and a feeling of illness, but he continued to go out. This period may be looked upon as the primary attack, and the graver illness as the relapse. On December 6, he got very wet, and, in the evening, shivered and sweated and had pains in his legs, but no fever. On the 7th, his temperature was 101°, and on the 8th and 9th, it varied from 102° to 104°, the pulse being always relatively slow. On these days he had several severe rigors, followed by sweatings, but the temperature did not fall after the latter. The abdomen became greatly distended and hard, and he vomited everything he took. The idea of intestinal obstruction was negatived by the fact that aperients opened the bowels well, though they did not relieve the distension. He passed no flatus except with the motions. On the 10th, the temperature being still high, the abdominal distension was greater, and not only was it more marked in the upper portion, but the outline of the stomach could be distinctly seen, beneath the abdominal wall, occupying an area much greater than the normal. He had been slightly delirious during the night, and the tongue was dry and brown. On the 11th, in the evening, the temperature was 104°, and the pulse only 84, (it was never over 90, however high the temperature). The heart, lungs and kidneys were normal throughout, but the fauces were generally swollen and purple. The abdomen was greatly distended, but the stomach could no longer be seen. The swelling was now greatest in the umbilical region, and I thought I could feel coils of distended bowel; in fact, it appeared that the gastrectasis had gone, and had been succeeded by an enterectasis. There was no actual pain, except from the sense of distension, and no tenderness, and, as the patient moved freely about the bed, peritonitis and, therefore, perforation of any part of the alimentary canal could be excluded, although the distension made it more comfortable for him to lie with his legs drawn up. The distension varied from time to time in degree, and, when it increased, the heart became extremely embarrassed. During the night the distension was less, and he slept well. On the morning of the 12th, the temperature was 102°, pulse 84, good quality. The abdomen was now fairly supple, which had not been the case since the beginning of the illness. He appeared to be going on well, but, on the 13th, in the morning, he again became greatly distended and very collapsed. During the night, the temperature was 103°, but in the morning 101°. This proved, however, to be the last attack, for, from the time of its subsidence, he made an uninterrupted recovery. The case seemed to be one of acute toxæmia, causing very high fever and marked abdominal symptoms. But the latter were not those of ordinary irritation (there being no diarrhœa), but rather of a successive poisoning of the pneumogastric and sympathetic nerves, producing a successive paralysis of the stomach and intestines, with consequent great distension of their lumina. The influence clearly came on in paroxysms, and the train of events well illustrates Dr. Althaus' dictum that in the abdominal form of influenza the symptoms are due to poisoning "of those portions of the nuclei and branches of the vagus, which supply the abdominal viscera, with occasional extension of the shock to the sphere of the splanchnic nerves." The collapse and the relative slowness of the pulse might be due to the action of the poison on the cardiac branches of the vagus, but, more probably, to the inhibiting action of the abdominal trouble on the heart. It may be added that, just before his illness, this gentleman had been in contact with at least two cases of ordinary influenza. I have not been able to find a case like this, but I see that Dr. Campbell Thomson states that one of his cases of acute gastrectasis was associated with an obscure fever.

The "treatment" of influenza of the gastro-intestinal tract must, of course, be expectant. It need only be said that, in view of the tendency to cardiac failure, strong measures must be avoided. In Dr. Frohlich's case (V. S.), a cold bath was given for the purpose of lowering the temperature, and it was followed by dangerous collapse. Both antipyretics and purgatives must also be used with caution. Intestinal antiseptics should be tried, and strychnia, digitalis, and alcohol will probably be needed.

INFLUENZA AND APPENDICITIS.

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Of the many surgical complications or sequelæ of influenza, none is of greater clinical interest and importance than appendicitis.

The immediate cause of appendicitis is always microbic infection, but experimental research has shown that it is not necessary to introduce virulent bacteria into the appendix to produce an inflammation. Given a slightly diminished resistance of the tissues, all the conditions necessary for an inflammatory outbreak are present. It is a well-known experimental and clinical fact that micro-organisms, which are incapable of infecting healthy tissue, easily invade tissue which is slightly altered by traumatism.

The frequent association of appendicitis with such constitutional diseases as rheumatism, and the occurrence of appendicitis in the form of small epidemics, have given rise to the theory, among many observers, of the existence of a pathological relationship between the general affections and inflammation of the appendix. In fact some writers go so far as to regard all cases of appendicitis as a local expression of a general infection. Reclus, while acknowledging the influence of general infection, holds that appendicitis is not a disease one and indivisible, but is produced by various causes, some being more local in character, while others are more general. Tripier concludes that, in appendicitis, as in the majority of intestinal lesions, a general infectious origin should be more frequently recognised, but thinks that the association may be more or less remote, since the previous disease may have given rise to one or more attacks of latent appendicitis, before causing an attack which manifests itself by the characteristic symptoms.

Howard Kelly thinks that the experimental proof of the susceptibility of the appendix to become the seat of a local process in a general infection should make us more ready to accept the clinical evidence.

Osler points out that, in all large epidemics, it has been repeatedly observed that the intestine is more or less seriously injured, the brunt of the catarrhal affection falling upon the gastro-intestinal mucous membrane. So that it is not to be wondered at if, in the appendix, which is already prepared, a more severe process frequently arises. It is certain that, during recent epidemics of influenza, an increase in the number of cases of appendicitis, and a frequent connection between the two diseases, has been noticed by many careful observers. Finney reports six instances in which the appendical inflammation appeared during, or soon after an attack of influenza.

Sonnenburg considers it to be an established fact that, during an epidemic of influenza, inflammation of the appendix is much more frequent. Abbé, quoted by Kelly, reports cases of appendicitis occasioned by influenza. In these cases, the disease seemed to be concentrated in the appendix, producing acute inflammation, with follicular ulcers and some hæmorrhage. It was accompanied by characteristic swelling of the regional lymph glands.

As further proof of the relation of appendicitis to a general infection, its frequent occurrence in epidemic form may be adduced. Gagnière[1] gives details of cases, which occurred at a time when appendicitis was prevalent during an epidemic of influenza, cases of appendicitis appearing during the influenzal period, and ceasing apparently coincidently with the epidemic. He had 900 cases of influenza under his observation, during an epidemic at Choisy-le-Roi, from December, 1898, to May, 1899, and the percentage of cases of appendicitis, occurring during this time, was about 1·3. He draws attention to the following points:—

1. That these cases of appendicitis were contemporaneous with an influenza epidemic.

2. That, before the commencement of the epidemic, he did not observe a single case, in spite of the number of patients examined (about a thousand), from May to October.

3. That, in some of the patients, the appendicitis was coincident with influenza.

He cites seven cases in support of his contention.

Bottomley[2] refers to the part that influenza may play in the causation of appendicitis. He thinks that it is more than probable that influenza may have some share in the production of the fluctuations in the increase, but is doubtful as to whether it can be blamed for the whole of it. Nevertheless, he appends a table, which shows that, with one exception, the years, in which the greatest number of cases of appendicitis was admitted to the London Hospital, were those in which the greatest number of cases of influenza were under treatment. He also thinks that there is a much closer correspondence between the number of cases of influenza under treatment, and the death-rate of appendicitis, and that the year (1899), in which there was the greatest death-rate for appendicitis, was also the year in which the largest number of cases of influenza was under treatment.

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[1] *Gaz. des Hôpitaux*, 1899, LXXII., 1181.

The causal relationship of the two conditions was first pointed out by French writers (Merklen, Larger, Gagnière, Florand). Faisans asserts that, so far from the association of appendicitis and influenza being exceptional, it may be regarded as the rule. Schulte[1] cannot endorse this view from the results of his own observations.

During the period between April, 1896, and March, 1903, he had an opportunity of observing four epidemics of influenza, and a series of cases of appendicitis, in a battalion of an infantry regiment. During these seven years, there were six hundred cases of influenza, and thirty-nine of appendicitis. A table is appended, showing the number of cases of the two diseases in each year. From January 6th to February 1st, 1899, there were 68 cases of influenza and two of appendicitis. The two latter cases were not complicated by influenza. There were 69 cases of influenza from January 3rd to March 1st, 1900. From January 10th to March 10th, 1901, there were 112 cases of influenza, and two of appendicitis, not associated with influenza. From December 5th, 1902, to February 24th, 1903, there were 156 cases of influenza, three of which were complicated by appendicitis.

Of the thirty-nine cases of appendicitis, which occurred during the seven years, two were relapsing cases. In ten cases, the appendix was removed by operation, two of the patients dying from peritonitis.

The influenza epidemics invariably appeared during the winter months, usually lasting from December till March.

The symptoms were disturbances in the organs of respiration, and also in the nervous system. Associated conditions, such as rheumatism, cerebro-spinal meningitis, orchitis, and myocarditis, were sometimes observed.

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[1] *Deutsch. Med. Woch.*, 1903, Vol. XXIX

In the first three epidemics there was not a single case of appendicitis which was complicated by influenza. The fourth and last epidemic differed very materially from the previous ones. It was the most severe, and continued the longest, and the cases required a far greater average duration of treatment. It also had a distinctive character, owing to its being associated with many affections of the alimentary canal. The symptoms at the commencement of the attack resembled those of slight typhus. The patient suddenly became ill, when in good health, and suffered from vomiting, severe pain in the abdomen, vertigo, headache, diarrhœa, high fever and prostration, but no cough or expectoration. In three or four days, the temperature went down, but the abdominal pain persisted, and, in some instances, became more severe.

It is further to be noted that nine cases of appendicitis were observed in connection with this epidemic, that is to say, during the period between November, 1902, and January, 1903. Five of these occurred before the appearance of a single case of influenza, and some of the patients had previously had several attacks. Of the patients suffering from appendicitis, who were admitted to the hospital at the height of the epidemic, some stated that they had previously had symptoms of influenza, or that they had appeared during the illness.

Goluboff is inclined to regard all cases of appendicitis, which come under treatment during the course of an epidemic of influenza, as complications of it. Schulte, however, considers that, while the conditions may be associated, they certainly originate independently of each other.

In the first three epidemics observed by him, there was no single case of appendicitis associated with influenza, and in the last epidemic, which differed characteristically from the preceding ones, only three cases of appendicitis were complicated by influenza. The inflammation of the appendix, in these three cases, he thinks may be explained as a catarrhal inflammation of the mucous membrane of the appendix, produced by the bacilli gaining access to it.

Adrian holds that the strongest argument against the condition being a sequela of influenza is that the influenza bacillus cannot be demonstrated in perityphlitic abscesses.

Franke considers that a relation between the two conditions is probable, in the sense that appendicitis is masked by the symptoms of influenza. He assumes the cases decribed as pseudo-appendicitis to be neuralgias affecting the region of the ilio-hypogastric nerve, and possibly also that of the ilio-inguinal nerve, and thinks that some of the cases, quoted by Florand and Faisans, come into this category.

Deaver thinks that influenza exerts a predisposing influence in the production of appendicitis, because of the intestinal lesions to which it gives rise. These changes affect the mucous membrane and lymphoid structures of the appendix; or drainage is effectually prevented by the swelling of the appendicular orifice. Or the influenza bacillus, gaining access to the appendix, may directly excite inflammation of this organ. He thinks that the prevalence of this disease, as a pandemic, may account in part for the great number of cases of appendicitis in recent years, and quotes Marvell's observations tending to confirm this statement. The latter writer shows that influenza and appendicitis have of late years increased almost *pari passu*.

In regard to the influence of influenza upon the operation for appendicitis, Follet[1] observed, during the course of an epidemic of influenza:—(1) That operations, especially laparotomy, and those requiring the administration of chloroform, might cause an attack of influenza, or a recrudescence or exacerbation of it. (2) That influenza is more severe in the presence of suppuration.

He refers to six cases in which laparotomy was performed, in all of which influenza appeared, or there was a notable exacerbation of it, immediately after the operation. Chloroform was administered in all these cases. It is possible that, in these cases, the symptoms were caused by (1) the action of the chloroform; or (2) the action of the operation itself. In three of the patients, cicatrisation was imperfect, and he raises the question whether influenza has any influence upon this process.

He also observed that patients, suffering from suppuration, were more liable to contract influenza, and evidently possessed less power of resistance to the affection.

[1] *Gaz. Méd. de Paris*, 1895.

CARDIAC COMPLICATIONS OF INFLUENZA.

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The cardiac symptoms, which may occur in influenza, contrast strongly with those of acute rheumatism. In the latter disease, acute inflammatory affections are common, and both the endocardium and the pericardium may suffer; while the myocardial lesions, though usually well defined, rarely occasion symptoms during the course of the attack. The cardiac danger is, on the whole, remote rather than immediate.

In influenza, on the other hand, endocardial and pericardial lesions are rare; and, while in a few instances they have been shown to be due to the specific bacillus, in probably the majority of cases they are the result of a secondary pyogenic infection. The danger in influenza is immediate rather than remote.

In an uncomplicated case of influenza in a healthy adult, special cardiac symptoms are rarely noteworthy. The pulse is, as a rule, somewhat less frequent than the degree of the pyrexia would warrant, and the arterial tension is well sustained; but, in cases, in which cardiac weakness from any cause is pre-existent, the pulse tends to become frequent, weak and irregular, even early in the attack, and the failure may quickly become extreme. Patients with a fatty heart, and alcoholics, bear influenza badly.

In healthy adults, too, the same sequel may follow, if the patient attempts to combat the disease without going to bed, and faintness, often of an alarming character, may occur in such cases on even trivial exertion, such as sitting up in bed. Fatal syncope is rare, and probably only happens after definite evidences of cardiac weakness have been manifested.

During convalescence, the cardiac action, as a rule, soon returns to the normal, but sometimes the pulse rate becomes rapid, or infrequent, or irregular, and the force of the systole is poor, and this is most likely to occur in persons with pre-existing cardiac flaws. It is important to recognise the cardiac fault at once, as symptoms may make their appearance only when the usual habits are resumed, and undue exertion has produced dilatation of the heart. As a rule, such symptoms manifest themselves in cases in which the acute attack has been severe, but they are not uncommon after the milder seizures, and the amount of exertion required to produce them may be almost absurdly trivial.

The symptoms are generally cardiac in character, palpitation and tachycardia on the slightest exertion being complained of; and feelings of faintness, or intense exhaustion, with profuse cold sweats, are common accompaniments; more rarely, dyspnœa on exertion, with perhaps slight swelling of the feet at night, brings the patient under observation.

In most cases, symptoms arising during convalescence are subacute, and occasion little danger, though much discomfort and inconvenience; but they tend to persist indefinitely, sometimes for months, and a sudden and fatal syncope occasionally supervenes.

The common cause of cardiac symptoms is myocardial weakness. The granular and fatty changes in the muscle cells, which are a constant feature in the acute infections as a whole, are always extreme in cases of influenza which come to section; and all the usual clinical evidence of muscle failure can be found on examination during life. Delayed failure, occurring in the later stages of convalescence, if not produced by over-exertion, appears sometimes to be due to neuritis of the cardiac nerves. I am only aware of one or two instances, in which such an inference has been justified post-mortem, but the remarkable resemblance between the cardiac symptoms in diphtheria and in influenza makes the assumption extremely probable. It is unlikely that degenerative changes should occur in the muscle cells after convalescence has been established, and it is well known that neuritis of the peripheral nerves, neuralgias, and indefinite cerebral symptoms not infrequently succeed an attack.

The tendency to cardiac strain, on slight provocation, must be kept in mind in the treatment of persons suffering from influenza. As everyone knows, a speedy recovery is most surely obtained by confinement to bed from an early period in the attack, and rest should be maintained afterwards for a longer period than is requisite in non-influenzal catarrhs. Undue slowness, or frequency, or irregularity, or weakness, of the pulse is a very special indication for continued abstinence from exertion, and a return to normal habits should always be accomplished gradually.

Cardiac weakness during the acute stages is best countered by the administration of strychnine and the diffusible stimulants, such as ammonia and alcohol, but the quantity of the latter which can be of value is more limited than is generally recognised ; six to eight ounces of whisky or brandy, well diluted, in the 24 hours, is probably the maximum dose. The special cardiac tonics, digitalis, etc., are generally less useful, but, in extreme cases, their assistance may also be invoked. Getting up to stool, etc., must be prohibited, if the weakness is great.

Cardiac weakness during convalescence, or succeeding an attack, demands continued care, and often persists for months. Strychnine, again, is the most valuable drug in these circumstances, but the hypophosphites, arsenic, and iron are also of service ; and a change of air to a warm and dry climate, with its enforced freedom from business or social worries, has often a very beneficial influence.

Cardiac murmurs, of functional origin, are often found on examination of the heart after an attack of influenza. If no special symptoms are present, they are of little importance, but care must still be taken of the general health. Proposals for life assurance should, in consequence, always be postponed for some time after an attack, so as to prevent the annoyance caused by delay in acceptance. If the necessity for assurance is urgent, a reference to the recent illness should always be made, so as to obviate the expression of an erroneous opinion as to the origin of the murmur.

THE NERVOUS SYSTEM IN INFLUENZA.

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It has frequently been said that, next to diphtheria, there is no acute malady after which disturbance of the nervous system is so frequent as after influenza, and none that has such varied nervous sequelæ. We may go even further than this, and scarcely be accused of exaggeration, if we assert that the prime sufferer in influenza is the nervous system, and that the large majority of its symptoms and sequelæ are the effects, direct or indirect, of the expenditure of the virus of Pfeiffer's bacillus upon nervous structures. Numerous and diverse as these may undoubtedly be, there is scarcely a disease of nerve cell or fibre that has not been ascribed to this most searching of diseases since the last great pandemic of the early nineties. We must, therefore, be on our guard against the facile error of debiting every *post hoc* as *propter hoc*, for, although it may be sometimes a justifiable euphemism to speak of a locomotor-ataxy or general paralysis as influenzal in origin, we must not overlook the essentially parasyphilitic nature of these diseases, and allow ourselves as well as the friends of the patient to be the willing victims of this delusion. No doubt, in many cases, the first symptoms of these diseases have appeared within a comparatively short time of a severe attack of influenza, and it is quite easy to understand how this disease, which exercises so profound a depressant effect upon the nervous system, may aid the development of symptoms of diseases which were previously latent. Although, in this manner, influenza may contribute to the production of tabes or general paralysis, it is probable that they are never, strictly speaking, influenzal sequelæ, and dependent solely, in their ætiology, upon the antecedent febrile attack.

It will be convenient to describe the effects of influenza upon the nervous system in the following three groups:—(1) The nervous symptoms appearing at the onset; (2) Complications arising during the illness; (3) Sequelæ.

The nervous symptoms appearing at the onset are well illustrated in the account of his own attack that a London physician gave in the *Lancet* of January, 1890. He states that he awoke on Boxing Day, 1889, feeling severe pains at the back of the eyeballs, frontal headache, and pains all down his spine. The pains were symmetrical, and slightly shifting in character, affecting all the posterior muscles, beginning at the neck and ending at the calves. There was entire absence of pain in the anterior muscles of the trunk and limbs; he felt extreme prostration and depression of spirits, accompanied by a feeling of nausea and pain in the epigastrium resembling sea-sickness. The severity of the symptoms lasted for two days, continued in less degree until the thirteenth day, when they disappeared completely.

After an incubation period of about two days, the onset of severe frontal headache, pains in the eyeballs, and in the back and limbs, with extreme prostration, are characteristic of the disease. Even before these acute symptoms appear, an unaccountable loss of muscular power may be noticed by the patient. A young man of 20, who was rowing hard daily in an eight-oared boat in training for the Lent races at Cambridge, in February 1890, noticed for two days, though he was feeling otherwise quite well, that he was unable to pull his weight in the boat, and to work so hard as he was accustomed to do, when suddenly, about eight in the evening, he was attacked by severe cramping pains in the back and calves and by such excessive weakness, that he was scarcely able to make his way back to his rooms and get to bed. This suddenness of the onset, of the collapse, and prostration in influenza is most striking, and Goodhart remarks that only in cholera is the collapse so sudden and continuous, flinging strong men down. Sudden faintness or syncope may be the first sign of the advent of the disease, as in the case of a man who was driving a trap along a country road, when in a moment he fainted and fell out of the trap on to the road. Recovering consciousness in a second or two, he was able to climb back to his seat and drive back home, where he was seen to be looking extremely ill and shivering, and was put to bed. He then went through a characteristic sharp attack of influenza, but could remember nothing about his drive from the time of starting.

The headache at the onset is nearly always frontal, and is often unusually severe, preventing all sleep, and racking the patient to such a degree with torture that he may become completely obsessed by the pain, and apparently delirious. The severity of this headache usually subsides after two or three days, though in some cases it is less noticeable at the onset of the illness, but may come on towards the end of the first week, when the fever and the muscular pains are beginning to subside. Frontal headache, accompanied by pyrexia, is not pathognomonic of influenza, since the two often usher in typhoid fever or small-pox, but, in addition to these symptoms, the sudden appearance of *severe pains in the back and limbs*, and especially at the back of the eyeballs, together make up a clinical picture that can scarcely be mistaken for anything else than influenza. The pains in the back muscles, calves and thighs may be very severe and shooting, resembling the lightning pains of tabes, or they may be more general; the patient complaining of a feeling of soreness all over, as though he had been thrashed with sticks. Occasionally the pains appear to be located in the joints, and may be mistaken for rheumatism, or they may seem to be in the bones, or in the chest, lumbar region, or in the testis. The neuralgic pains about the thoracic or abdominal viscera may simulate various disorders of these organs, and may be mistaken for pleurisy, pericarditis, stone in the kidney, or bladder, hepatic or intestinal colic; these severe pains rarely last more than two or three days. The onset of the acute symptoms is almost always accompanied by a certain amount of pyrexia, the fever usually ranging from 101° to 104°, though all variations may be met with; temperatures of 105° are not very uncommon, and even hyperpyrexia may occur. It is undoubtedly true that a certain number of cases of influenza may show no pyrexia at all, although the other symptoms may be more or less characteristic of the disease. It has often been remarked that the rate of the heart-beat in influenza is disproportionately slow to the fever that may be present, a pulse of 80 or 90 being not uncommon with a temperature of 101° to 102°. The tongue is usually large and indented, and soon becomes covered with a thick creamy fur, while the breath becomes peculiarly fœtid. *Sweats* too, often severe and drenching, are not uncommon during the course of the fever, and it has been remarked that the perspiration, like the breath, has a peculiar sickly odour. With the sweats, *rigors* are likely to occur, and a shivering fit may even mark the onset of the disease. A peculiar feeling of giddiness and light-headedness may be a most distressing early symptom, and is likely to be associated with an intense feeling of mal-de-mer, retching, and vomiting. The vomiting may resemble sea-sickness in its noisy, spasmodic character, and may be much intensified by food.

In some attacks, repeated fits of sneezing, with the onset of severe coryza and running at the eyes, may usher in the illness. The sneezing and running at the nose and eyes may resemble hay-asthma, while, in some patients, there may be spasmodic attacks of wheezing and dyspnœa resembling ordinary spasmodic asthma, with great respiratory distress and lividity of the face. The precordial sensation of oppression may be so distressing as to resemble angina pectoris, and the patient may complain of a feeling as if the heart would stop at any moment. Suddenly as the disease may make its appearance, its course is apt to be comparatively protracted. The severity of the pains in the back and limbs, the fever, and the frontal headache, are usually much diminished after two or three days, and in the slighter cases these symptoms may completely disappear in this time. A most characteristic symptom of influenza is, however, likely to be left behind, an extraordinary muscular weakness and depression of spirits, which frequently last from one to two weeks. In the favourable cases, which form the majority, there may be no further complications, and the intense lassitude and depression clear up in a few days, and the illness is then over. In a great number of instances, however, the story is not so simple, and the tale of symptoms is lengthened by a formidable list of complications and sequelæ that may arise in connection with the disease.

Complications.—In addition to the slow heart and fainting, with sudden syncopal attacks that may occur at the onset, the action of the heart may later become irregular or extremely rapid. A permanent degree of dilatation of the heart with irritability of action and tendency to tachycardia may follow this condition, and even Graves' disease with exophthalmos and goitre may ultimately develop. Other vascular lesions in connection with the nervous system are cerebral thromboses. Right hemiplegia, with motor aphasia and agraphia, has been recorded, due no doubt to thrombosis of the middle cerebral artery; sudden onset of blindness has also been observed, clearing up later and leaving permanent right hemianopia, due to a vascular lesion in the occipital region, probably a thrombosis, causing a patch of softening in the calcarine cortex on the left side. The headache may fail to improve after the first two or three days, becoming worse with the development of symptoms of *meningitis*. The pain in the head then shifts somewhat in position, becoming chiefly occipital and vertical. The pain is apt to spread down to the back of the neck, which becomes stiffer and more tender, with perhaps slight head retraction. The optic discs become blurred and swollen, and there may be also some symptoms of spinal meningitis, with severe pains in the limbs and tenderness along the nerve trunks, drawing up of the legs, and increased or absent deep reflexes. The cranial nerves as a rule escape, with the exception of convergent strabismus from sixth nerve weakness, and some difficulty in swallowing from pharyngeal and palatal palsy.

Amongst the most important of the complications are the *ocular phenomena*. *Optic neuritis*, with choked discs and retinitis, was not unfrequently seen in the epidemic of the early nineties. It is apparently a toxæmic symptom and does not indicate the presence of meningitis, in which condition the papillitis rarely amounts to more than blurring of the edges of the disc, with swelling of one or two dioptres. *Influenzal retinitis* is peculiar from the frequent presence of starry, fan-shaped groups of white spots in the region of the macula, and between the macula and the optic disc. Vision may be seriously damaged by *retro-bulbar neuritis*, due to a hæmorrhagic inflammation of the macular bundle of fibres in the centre of the optic nerve, producing central scotoma with great diminution of acuity of vision and loss of discrimination of colours. Embolism and thrombosis of the central artery of the retina have also been recorded, causing sudden blindness, or great defect in sight if only a branch of the artery is occluded. Irido-choroiditis, with its consequent injury to vision through occlusion of the pupil, vitreous opacities, and adhesions of the iris to the lens capsule may also be serious. Comparatively common were varying degrees of *ophthalmoplegia.* The most frequent of these is loss of accommodation, no doubt due, as in post-diphtheritic neuritis, to a localised paralysis of the ciliary muscle. Mostly the pupils too become sluggish in their reaction to light when there is loss of accommodation, and in a very few cases the reaction to light is completely lost. In addition, there may be ptosis, paralysis of any of the external ocular muscles, or even complete third nerve palsy. Goldflam recorded a case which developed complete bilateral ptosis, with total immobility of both globes, following fourteen days after influenza. The pupils were normal, but there was weakness of the soft palate with difficulty in swallowing. Gillet de Grandmont published a somewhat similar case with bilateral ptosis and complete external ophthalmoplegia, with weakness of accommodation and sluggish pupils. Other severe symptoms present were double optic neuritis and pronounced somnolence, with weakness of the extremities. Oppenheim similarly has observed double ptosis and bilateral ophthalmoplegia, with weakness of accommodation following influenza. In addition there was difficulty in articulation and in swallowing, with slight fever, severe tachycardia, and paresis of the extremities. This case recovered in five weeks.

Gutmann recorded a case in which there developed early a complete third nerve palsy, with the eyeball turned outwards and motionless, and complete ptosis; the pupil was dilated and inactive to light. Later on the external rectus also became paralysed, and the superior oblique as well, the eyeball being fixed. Ultimately the ocular paralysis cleared up, and the reaction of the pupil to convergence and accommodation returned, though it remained inactive to light. These examples of severe opthalmoplegias complicating influenza are somewhat rare, but the slighter forms of paralysis of single muscles, and especially of loss of accommodation, were fairly common during the epidemic. It will thus be seen that the ocular paralyses which occur in influenza last longer, and are more severe than the post-diphtheritic forms of ocular paralyses. I can find no record of any autopsies on these cases, and Wilbrand suggests that they are due to a hæmorrhagic polioencephalitis, affecting the region of the third fourth and sixth cranial nuclei in the pons, *polioencephalitis inferior.* Leichtenstern first described encephalitis as a special complication in influenza, and Goldflam has recorded a case of polioencephalitis, superior and inferior, with acute anterior poliomyelitis, followed by death, after influenza; that is to say, an acute hæmorrhagic lesion of the grey matter of the cortex, of the pons, medulla and of the anterior horn cells in the spinal cord, occurring at the same time as a fatal complication of influenza. *Acute encephalitis*, or polioencephalitis superior, is usually ushered in with a convulsion, which may be unilateral; or in children, with a series of convulsions, followed by prolonged coma lasting perhaps several days. When occurring as a complication of influenza, it is likely to occur between the second and seventh days of the illness. It is a toxæmic infection of the meninges and pial vessels with Pfeiffer's bacillus of influenza, and is likely to be accompanied by signs of meningitis also. The grey matter will be found to contain minute points of hæmorrhage, and the nerve cells swollen and exhibiting signs of chromatolysis. Lumbar puncture in these cases may be successful as a means of demonstrating influenza bacilli in the cerebro-spinal fluid withdrawn. If the case recovers, the symptoms, resulting from the acute cerebral lesion, will depend upon the part of the cortex that has suffered; if the Rolandic area has borne the brunt of the attack, hemiplegia will result, if the frontal lobes have been the chief site of the mischief, then permanent mental impairment will ensue; if the occipital cortex has been attacked, then impairment of vision will be the result; at first probably complete blindness, which may be in rare cases permanent and complete, but if one occipital lobe has suffered more severely than the other, then the visual field is likely to clear up on one side, leaving behind a permanent hemianopia; sometimes the whole visual field may recover, as I have seen after complete blindness lasting for two weeks. Further evidence of the poisoning of the cerebral centres by the influenzal toxin is afforded by the peculiar dazed feeling and apathy, with extreme depression and bad dreams, that often occur as initial phenomena in the disease.

Hysteroid *convulsions* may first draw attention to the patient's serious condition, and they may be followed by fainting, stupor, or even a cataleptic state. Grasset and Ranzier record the case of a French soldier, æt. 28, who had a hysteroid fit on the second day of his illness, the arms and legs being acutely flexed, lasting half an hour without loss of consciousness, but involuntary urination afterwards. This was followed by temporary right hemianæsthesia, which after a short time cleared off except from the arm. He was completely unable to move the wrist or fingers, and there was complete analgesia of the hand up to a circular line above the wrist, though tactile sensation was unimpaired. There was also loss of the pharyngeal reflex. As an instance of *catalepsy* the following case may be cited :—A man was seized with influenza, with shivering, abdominal pains, vague pains all over, constipation, anorexia, restlessness, and temperature 103°. After four days the symptoms subsided sufficiently for him to be able to get up. He then suddenly lost consciousness, and was found lying on the bed apparently senseless, with his eyes open, the face pale, and pulse weak, 120 per minute. There was generalised tonic rigidity of the muscles, so that the limbs would remain in any position in which they were placed, so-called lead-pipe rigidity ; this state lasted for two hours, and was followed by recovery. At the time of the epidemic, in 1889-90, there was considerable scare in Switzerland over the reported appearance of "*Nona*," or a condition of apparent death. It is a condition of extreme somnolence, or deep trance, simulating death, but it is not peculiar to influenza, and has been seen in cerebro-spinal meningitis. A case of apparent death of a man was reported in the *Lancet* for 1890. After due consultation a certificate of death was signed ; an hour before the time fixed for the funeral, signs of recovering consciousness were noticed by the friends, and, as the result of efforts to restore animation, he recovered to the point of recognising those around him, but he again lost consciousness and died.

The following case of *trance* after an attack of influenza was reported by Dr. Nathan Raw. A woman, always hysterical, and a deep student of the Bible, had a severe attack of influenza, with excruciating pain in the head, and was quite prostrate in mind and body for two weeks. She then fell into a deep sleep, and dreamt that she heard a voice saying to her, "You are dead." She lay quite helpless and flaccid for ten days, and could not be roused by any means, nourishment being given by a stomach tube; she afterwards recovered rapidly. Young infants, if seized in the grip of influenza, are more likely to be overcome by the toxæmia, and lie in a somnolent state. This may be compared with the different way young infants react to acute lobar pneumonia as compared with adults. Instead of the active delirium and sthenic symptoms exhibited by the latter, the babe lies in a drowsy condition as though drugged. So, in influenza, they may lie in a prolonged somnolent condition throughout the febrile attack for a week or ten days, and then recover rapidly.

Certain visceral symptoms, occurring early in the course of influenza, may be attributed to disturbance of the nerve supply to these organs by the infection. Such are the neurotic polyuria and the watery diarrhœa that are sometimes met with. Constipation is, however, more frequently present. Spinal disorders, that may complicate influenza, are acute anterior poliomyelitis, disseminated myelitis, ascending myelitis, Landry's paralysis and herpes zoster. Spastic paraplegia has also been recorded as a sequel, but unless it is the result of an acute attack of myelitis occurring as a complication, it is doubtful if the spastic paralysis can rightly be included as a direct result of influenza. Jolly saw a case of poliomyelitis in a lady, which came on three weeks after influenza with pains and paralysis of the left arm, and later of the left leg. Fiessinger records a case of polioencephalitis inferior in a young girl of 18, who, after the usual symptoms of influenza, developed, on the fifteenth day nasal speech and difficulty of swallowing, with opisthotonos. The motor and sensory power of the extremities was good; pulse 160, followed by death; no autopsy. Bulbar crises, resembling the attacks seen in children with diphtheria, with spasm of diaphragm or glottis and inability to swallow, have been met with, and may be rapidly fatal.

Myelitis, complicating influenza, is usually cervical and accompanied by meningitis, or it may be scattered in separate foci over a large area of the spinal cord. Paraplegia is the prominent symptom, at first flaccid, and, if the patient survives long enough, spasticity develops later. Incontinence of urine and fæces, anæsthesia of the lower part of the trunk and lower limbs, sometimes muscular wasting in groups of muscles distributed according to spinal segments and not to their peripheral nerve supply, and rapid onset of severe bedsores indicate a severity of the spinal injury from which recovery rarely occurs. Idiopathic tetanus has more than once been recorded as following a few days after an attack of influenza, but precisely in what way the influenzal attack assists the advent of the tetanus bacillus to the nervous system, we are completely ignorant. Féréol, amongst others, has recorded a case of *ascending myelitis* complicating influenza. This is invariably fatal, starting with loss of power in the lower extremities and anæsthesia of the gluteal region, spreading up the legs and trunk, so that by the end of a week the whole of the lower portion of the body below the arms may be completely anæsthetic and powerless, respiration being carried on entirely by the diaphragm. The reflexes, superficial and deep, are lost from the commencement, and if the patient survives for more than a week, reaction of degeneration in the muscles of the lower extremities will be found on electrical testing.

Landry's paralysis, so called, may also occur, differing from ascending myelitis clinically in the absence of any marked degree of anæsthesia or muscular wasting. and little or no involvement of the sphincters. In their morbid anatomy, however, there is a certain resemblance, though occasionally a case of Landry's paralysis may survive. In cases of ascending myelitis, the whole cord, more especially in the sacral and lumbar region will be found swollen, more or less diffluent on section, with small hæmorrhages scattered throughout both grey and white matter. The pia-arachnoid covering the cord is also involved in the inflammatory process, and the condition is really a spreading meningo-myelitis. In many of the fatal cases of Landry's paralysis, which have come to autopsy, either nothing abnormal has been found, or slight changes of a chromatolytic nature of the nerve cells and some swelling of the fibres. In some others, though the cord and pia-arachnoid showed very little change, the dura mater covering the cord was found inflamed, and the seat of a micrococcal infection, which suggested that, in some cases at any rate of

Landry's paralysis, the spinal theca is the part specially attacked.

As to whether, in the cases of this affection complicating influenza, Pfeiffer's bacillus may similarly invade the spinal dura and produce these symptoms, we have no knowledge ; in all probability, the bacillus of influenza is capable of producing a spreading meningo-myelitis, which may be either slight or severe, the less severe forms having been described clinically as cases of Landry's paralysis complicating influenza.

Herpes zoster, as is only to be expected, has frequently occurred during the course of influenza. Zoster has been shown to be an acute infection, resembling in character acute anterior poliomyelitis. In the latter, it is the anterior horns that suffer almost exclusively, while in zoster the stress is borne by the posterior root ganglia, in which hæmorrhages and acute degeneration and swelling of the nerve cells have been found. Most commonly it attacks one of the dorsal roots, so that pain and redness of the skin round one side along the course of an intercostal nerve is the first symptom, followed by a crop of vesicles mapping out the distribution of the posterior root which is attacked. When, as is usually the case, it is one of the dorsal roots that suffers, the vesicles are found in two positions especially, behind, over the posterior division of the nerve, and in front, for four or five inches along the intercostal space up to the middle line. Zoster leaves permanent scars, but unless the patient attacked by it is over sixty, it leaves no other permanent effects. In old people, however, it may be followed by the most persistent and intolerable neuralgia, which nothing short of division of the posterior root internal to the ganglion will cure. Patients have indeed been driven to suicide by the unbearable severity of the pain. Zoster has been met with in influenza over the neck and occiput, affecting the higher cervical posterior roots, and this has been accompanied by facial paralysis on the same side. The Gasserian ganglion being precisely comparable anatomically with the spinal posterior roots, it is not surprising that herpes zoster sometimes occurs over the face and forehead along the distribution of the fifth nerve. Herpes frontalis, or ophthalmicus, is a special form which attacks the first division of the fifth nerve only, and may produce serious ulceration of the cornea in addition to the outcrop of vesicles over the forehead and side of the nose.

In addition to the cerebral and spinal diseases already referred to, which may arise in the course of an attack of influenza, the peripheral nerves also may suffer, and severely. These may be affected either as a local neuritis, or as a peripheral multiple neuritis, or by neuralgic pain only.

Local neuritis may occasionally attack certain cranial nerves, such as the fifth or seventh, causing weakness of the jaw muscles with pain and anæsthesia over the face, forehead, tongue, and inside of the cheek, when it is the trigeminal that suffers, and paralysis of the facial muscles or Bell's palsy, when the seventh is damaged. The eighth cranial nerves have also been known to suffer in influenza, causing a peripheral nerve deafness. This is to be distinguished from the deafness that may result from the middle ear disease and mastoid trouble, which follow an initial abscess in the ear, with severe pain, that not uncommonly is an initial symptom in an attack of influenza. The ocular paralyses have already been dealt with, some of which, especially the isolated muscular palsies of external or internal recti, or loss of accommodation, are due to neuritis of branches of the third nerve or the abducens, while the more severe cases of total ophthalmoplegia are much more probably due to a hæmorrhagic lesion in the floor of the aqueduct, known as polioencephalitis inferior.

Neuritis of the brachial or lumbar plexus on one side producing brachial or sciatic neuritis has been fairly common after influenza.

Brachial neuritis develops with rapid onset of intense pains down one arm from shoulder to fingers, made worse by the slightest movement, and entirely preventing sleep. It is due to a perineuritic inflammation of the sheaths of the nerves entering the plexus, a condition which also may occur in gouty persons, or from rheumatism, or exposure to cold. In a severe case, the arm becomes swollen, œdema appearing on the hand and wrist; the skin becomes shiny, and ultimately the muscles waste and trophic lesions appear on the skin and nails, glossy skin, wasted finger pads, and stiffness of the joints, especially of the fingers and wrist, form periarticular adhesions. In less severe cases there may be complete recovery after about a month.

Sciatic neuritis, or sciatica, is a similar process affecting part of the lumbar plexus, causing severe pain down the back of the thigh and in the leg and foot. The chief site of the pain is often in the ankle, with tenderness along the sciatic nerve on pressure in the mid-gluteal region, back of thigh, or popliteal space. With severe attacks, swelling of the foot and ankle and even the appearance of bullæ on the skin are found. Movement, as in brachial neuritis, intensifies the pain, and a severe attack may similarly be followed by trophic lesions of skin and muscles, though this is, on the whole, a more uncommon result in the leg than in the arm.

There is scarcely a nerve twig, or nerve trunk, that has not been known to suffer from influenza, with resulting local tenderness and trophic symptoms of skin or muscle persisting for variable periods. As was said in the case of herpes zoster, so with the innumerable post-influenzal nerve inflammations; in young people the trouble generally clears up entirely, leaving behind no permanent ache nor pain, while in older persons, roughly in proportion to their weight of years, influenzal neuritis is more difficult to get rid of, and frequently leaves behind most intractable neuralgia and tender points, even though there may be no more definite signs of trophic disorder of muscle, skin, or joints.

Peripheral Multiple Neuritis when severe is one of the most formidable complications that occur in the course of influenza. It is one of the later complications to appear, generally three weeks after the onset of the illness, but it may be as long as six weeks. It is really a complication rather than a sequel, as the morbid process is a toxæmic parenchymatous degeneration of the nerve fibres themselves, thus differing from the perineuritic inflammation of the sheath of single nerves or nerve plexuses, which is found in brachial neuritis, or sciatica, or other forms of local neuritis. The multiple neuritis occurring after influenza is similar in character to that which occurs after other toxæmic infections, such as diphtheria, varicella, measles, pneumonia, typhoid, beri-beri, septicæmia, and other infections of microbic origin, or the multiple neuritis that follows chronic poisoning with alcohol or arsenic. It is, therefore, unnecessary to describe the form of the neuritis at length, but to mention its chief features and the points of difference, which will help to distinguish it clinically from the multiple neuritis due to these other toxic causes. Considerable pain may mark its onset, with shooting pains in the limbs and general nerve tenderness, and weakness of the extremities, especially of the feet and ankles. The form of the paralysis is rather more motor than sensory, and muscular wasting with reaction of degeneration may ensue. Paralysis of the lower extremities, loosely described as paraplegia, is probably in most cases due to multiple neuritis. In a few cases, dangerous complications from cardiac and respiratory paralysis may be met with, but they are far less common than in diphtheria or beri-beri, while the sensory changes, anæsthesia, etc., are usually much less marked than in the alcoholic form. Bilateral facial paralysis may occur as part of a multiple neuritis due to influenza, as it may from alcohol, or the rare form of rheumatic general multiple neuritis. As in other forms of multiple neuritis, there is no sphincter involvement unless there is, at the same time, a patch of myelitis developed. Influenzal multiple neuritis is much less likely than diphtheria to commence with cranial nerve paralyses, though the ocular and other cranial nerve paralyses, when they do occur, are more severe than in diphtheria, but are due to local neuritis or a hæmorrhagic polioencephalitis, and are not a part of the multiple neuritis.

Peripheral neuritis in influenza, though a severe, is rarely a fatal complication, and requires treatment on the usual lines of multiple neuritis, with rest in bed so long as there is any sign of the neuritis increasing, and later massage, exercises, strychnine, and galvanic treatment.

Neuralgia has already been mentioned as occurring after herpes zoster, or local neuritis of single nerves or of a plexus. Trigeminal neuralgia may date from an attack of influenza in which there were at the time no signs of actual neuritis of the fifth nerve. Neuralgia differs anatomically from neuritis in the absence of any discoverable lesion in nerve-cell or fibre in the former, but the term is often used loosely, by expert physicians as well as by the laity, for the severe lancinating pains radiating along the course of a nerve trunk and its branches, which may be reflex pains from irritation of a single twig, as in a carious tooth, or the after pains left behind after a severe neuritis, as in herpes zoster, brachial neuritis, or sciatica. The treatment of these neuralgic pains must be attempted on general lines, though perhaps post-influenzal neuralgia, more than other forms, may be controlled by quinine.

Many SEQUELÆ have followed influenza, some of which, more than others, are directly dependent upon the disease. One which is perhaps peculiar to influenza is the prolonged *loss of power of smell and taste*, which often lasts for many months, or a year, after the acute illness. It is usually the sense of smell alone that is really affected, the power of appreciating salt, sweet and bitter, being retained, but all flavours and the taste of food are not distinguished on account of the loss of smell, which is probably due to a neuritis of the olfactory nerves. I have known it permanently lost after influenza.

Progressive bulbar palsy has also been recorded, with paralysis of the vocal cords, palate and pharynx, and tremors of the lips and eyelids, beginning acutely a few weeks after influenza, and steadily progressing.

Myasthenia gravis, too, has been recorded by Remak, in a girl of twelve, following five months after influenza, beginning with bulbar symptoms and ending with attacks of intense dyspnœa, death occurring in four months.

Epilepsy has been noticed to recur during an attack of influenza after years of intermission, and to continue in an aggravated form. This is probably due to a certain degree of cortical damage from encephalitis, as epilepsy is a common sequel to this dangerous cortical disease.

Migraine too may become aggravated in frequency and intensity.

Paræsthesiæ of all sorts and descriptions have been described as sequels, including pins and needles sensation, vague pains, numbness, hyperæsthesia, etc.

Infective polyarthritis and *diabetes*, too, may be formidable legacies of influenza. The polyarthritis, though not a nervous complication, may be mistaken for paraplegia, or for multiple neuritis, with its subsequent joint fixations from periarticular adhesions.

Lastly, we may mention the extraordinary effect that influenza often produces upon the mental functions, though this subject is being dealt with at length in another article. *Loss of memory*, and an intense *inertia of mind and body* may last for weeks and months after the acute illness, and all the well-known symptoms of grave neurasthenia, as well as more grave mental disorders, may remain and become permanent. This is in all probability due to the severe toxæmia from which the cerebral cortex has suffered during the infection; although it may not in all cases produce lesions of nerve cells and the grey matter that can be recognised as due to an encephalitis, yet the function of this grey matter may be permanently damaged by the poisoning, though modern methods of staining are unable to recognise the changes. This condition is in fact similar to the grave neurasthenia that may result after any severe mental shock, such as the complete upset of nervous balance that may result after escape from drowning, as has been frequently remarked in the case of the survivors from the ill-fated battleship *Victoria*. Many of these, previously strong men, with little or no family tendency to neuropathy, have been totally unable since to continue in the navy, and even the sound of splashing water may completely upset them. The same nervous breakdown has been noticed in the survivors of the fearful fire at the Colney Hatch Lunatic Asylum, many of whom, though removed to other asylums, still cannot bear the sight of a lighted match. It is, as might be expected, in those whose families show a heredity of insanity, or other nervous ailments, that such grave and permanent neurasthenic breakdown is more likely to occur.

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THE PSYCHOSES OF INFLUENZA.

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In this chapter, I am concerned especially with the psychoses as distinguished from the neuroses of influenza. In some respects, the two conditions overlap, for encephalitis, epilepsy, coma, and apoplexy, when they occur, as a result of the influence of the toxins or of focal hæmorrhages, are displayed by symptoms which, both in form and acuteness, affect the actual mental condition. Inasmuch as "neuritis and nerve disorders" form the subject of a special article in this collection of the hydra-like symptoms and after-effects of influenza, I propose to confine myself to the strictly mental as distinguished from the nervous states which accompany the disease, or which follow as sequelæ.

The discovery of the Pfeiffer bacillus in 1902, two years after the great invasion of Influenza, in 1889–90, was epoch-making in the history of the disease, removing it from the false basis of miasmatic or paludal poisons, and firmly establishing its bacteriological origin, thus bringing the classification of the mental symptoms under the head of the toxic insanities; a group of a very definite kind, and one which is daily becoming more and more exploited in giving us true ideas as to the causation of many hitherto obscure insane conditions. In what has been termed the delirium of acute pneumonia, in the early stages of variola, in some forms of syphilis, in typhus and enteric fevers, in diphtheria, and in epidemic cerebro-spinal meningitis, we note two classes of symptoms, first those due to the development and action of the organism itself, and secondly, those which follow the production of toxins, or which depend upon the mechanical results of focal hæmorrhagic complications; and the same dual condition may be seen in influenza, where there may be a sudden apoplectiform onset, either at the very beginning or at the height of the attack, or even a condition of coma, Jacksonian epilepsy, etc., or, secondly, a purulent encephalitis or abscess, either from otitis or from suppurative processes in the frontal and nasal sinuses. We shall see that, in the same way, the strictly mental symptoms may occur at the onset of the disorder, and may be of a foudroyant, nerve-storm character, or may appear later on in a still dangerous, if less notably excited condition, whilst, as in most of the toxic insanities, the tendency towards recovery is great.

In the great pandemic of 1889–90, the chief complications were shown in the respiratory organs, and the nervous system was distinctly second in order of attack, but yet there was a clear and prominent mental complication in many cases. As, however, the disease was to most practitioners a new one, and as the severity and general distribution of the complaint were very extensive and time-absorbing, and the microbic nature of the disease was only suspected and not proved, relative statistics of the different complications and groups of symptoms were only imperfectly made in this country; and it has only, in later years, been possible to make more accurate observations, and to place the nervous displays in their true category. It has been the custom to speak of the pulmonary, or the gastric, or the nervous form of influenza, according as one or the other set of organs was chiefly affected, but I am inclined to think that there has always been a fair proportion of nervous and psychic implication, and that this proportion has always been about the same in different epidemics, though, for the reasons given above, greater attention has been possible in the later and less extensive epidemics, hence the amount of nervous and psychic affection, though still considerable and important, has been exaggerated. During the great pandemic of 1889–90, I was in charge of more than 2,000 patients at one of the London Asylums, and was able to notice the effect on people already insane. For a long time, none of the patients took the disease, but after the attendants had become affected in large numbers the patients followed, and though I endeavoured to compare the percentage of those affected with that of those who remained free, it was found impossible to obtain accurate figures; certain it is, however, that the effect upon the already damaged nervous systems of the patients was not material, indeed I do not remember that any noticeable accession or diminution of the psychic symptoms occurred in any of the patients, except perhaps that the demonstrations in some of the more violent were masked during the few days of the attack, to reappear soon in their previous intensity. In no instance did the course of influenza appear to have a curative effect upon the insanity, as might have been expected from what is seen in some cases, in which the accession of an acute febrile disorder, such as scarlatina, may be followed by the cure of the insane state. If then it was not possible to obtain trustworthy statistics in an institution, where all the essentials for determining them were to hand, it can be easily concluded how much more impossible it was to obtain correct statistics drawn from general practice, especially when, in the absence of determination of the presence of the bacillus of Pfeiffer, there was often great doubt as to whether the patient was suffering from a severe cold or from a mild attack of influenza.

Opinions vary as to the effect of hereditary and other neuropathic influences on the production of psychosis during, or immediately after, an attack of influenza. According to some authors, the presence of hereditary taint or of nervous degeneration had no effect in making the subject more liable to post-influenzal insanity; but Mohr and Krapelin say that the probability of an attack does depend upon heredity or acquired psycho-pathic condition. The difficulty of obtaining correct histories of hereditary taint of insanity makes it, in this country, impossible to settle the question as to the effect of predisposition in determining an attack during or after the influenzal period; but the following observation which I made at the time may be mentioned, viz.:—That the existence of acute and chronic insanity did not render those, who were in that condition, more liable to an attack of influenza, because, when the epidemic was in full swing, the number of the already insane, who were attacked, was proportionately much less than among the sane population of the asylum.

Considering the small number of persons admitted to asylums (about 6·7 per cent. of the admissions), whose insanity was directly traceable to influenza, whilst hundreds of people (in whom or in whose family, though a psycho-pathic condition of some degree existed, yet no attack of insanity supervened) were victims to the disease, it would seem probable that Kräpelin's deduction must be received with caution. I am speaking now of distinct attacks of insanity, and do not include the simple depression, which is a frequent accompaniment of influenza just as it is of other acute diseases, and which is traceable, partly to panic and fear, and partly to the weakness following an acute febrile condition attended with high temperature. To put it in another way, there does not appear to be very trustworthy evidence that a person, in whose family insanity was strongly marked, would be more liable on that account to the "nervous" rather than to the "gastric" or "respiratory" type of influenza, though this question must still be considered an open one.

It was remarked by Nothnagel, in 1890, that "influenza produces a specific nervous toxin, which, by its poisonous action on the cortex, calls forth these psychoses," and there is no doubt that depression in the affective tone, a feeling of incapacity, of good-for-nothingness, was a very common symptom, but in the majority of cases this feeling passed off in a short time and left no bad consequences, though in others, perhaps from neglect of precautions, a more serious and real condition of true melancholia followed. Thus in Paris, it is stated, during the pandemic of 1889–90, the suicides increased 25 per cent., a large proportion of the excess being attributed to the nervous prostration caused by the disease. It is common knowledge that, at the outbreak of the epidemic, and for some time after, people did not take their condition seriously enough, they went about and occupied themselves in business and pleasure as usual when they ought to have been in bed, and so great were the exigencies of household and public duties that extra work was thrown upon those who were little able to perform it ; it is, therefore, not to be wondered at, that what was merely a primary depression of little significance, if given a chance of rest, became under stress a condition of real danger. Now that people understand better the danger of treating an attack of influenza with contempt, we are in a better condition for enforcing necessary precautions, and there is less danger of the already-taxed nervous system being allowed to suffer from protracted stress. If "stress" is, as appears most likely to be the case. the one important factor in the production of insanity nowadays, how banal it must be to allow people to keep at work under it, with an organisation already impaired by the influence of a known bacillus and its toxins! Small wonder is it that, in such circumstances, mere depression devolved into suicidal melancholia! It was probably the virulence of the great pandemic of 1889–90, and our medical and social unpreparedness for it, which caused the psychoses to assume the proportion to which they undoubtedly attained, but with the better knowledge of the disease at which we have arrived, there is every hope that bad results, as regards the development of insane conditions, will be much minimised.

Just as the form and the onset of influenza simulated, in some instances, diseases of a very different character, rendering accurate diagnosis difficult and uncertain, so in some rare cases was there a prodromal stage of one or more days of acute febrile dementia of the melancholic type, preceding the influenza, and marking the true nature of the complaint. In times of epidemic too much caution cannot be displayed in estimating the true nature of these early and more conventional symptoms, for to act upon them merely as signs connoting insanity, and to place a patient so affected under certificates because of his violence or stupor might be a very disastrous proceeding. It is better to remain in an attitude of expectant attention, for in a short time the true nature of the symptoms will be made manifest. There is no doubt that cases of this kind are due to the action of the bacilli, for, in 1892, Pfuhl saw simple meningeal congestion, with abundance of ventricular fluid and abscess of the brain, and Nauwerck reported hæmorrhagic encephalitis and true suppurative cerebro-spinal meningitis with Pfeiffer's bacillus well in evidence. The symptoms reported were headache, body pains, general lassitude, vertigo, delirium, and violent action, loss of consciousness, etc., and the bacillus was demonstrated in the cerebro-spinal fluid drawn off by lumbar puncture. With the exception of the inflammatory nervous conditions here alluded to, which must be attributed to the invasion of the influenzal bacilli, or of the micrococci associated with them, it does not appear that the presence of the bacillus has been determined in the usual forms of psychosis associated with influenza. I have consulted some eminent bacteriologists, and have searched the literature on the subject, but have not been able to find any account of the presence of the Pfeiffer bacillus in the blood, or other fluids, in any of the cases in which purely mental symptoms occurred during the course of, or directly after, the influenzal attack.

Nothnagel insists that the cause of the functional disturbances is the toxins produced by these micro-organisms, whilst others say that these conditions are not due to toxins, but are just the after effects of the acute and exhausting disease, and they point to the fact that the toxin of the bacillus has not yet been isolated. The bacilli Pfeiffer are the smallest rods yet cultivated, they thrive with difficulty, and their period of viability is about fourteen days, so that, unless they are looked for in the early stages of acute nervous and mental affections in influenza, they are not likely to be found. The toxins, however, are intense nerve poisons, and without doubt have been the direct cause of numerous polymorphic diseases of the nervous system, such as neuralgias of all kinds, hyperæsthesias of the skin, loss of sensations of smell and taste, and of paralyses due to neuritis, such as nuclear paralyses in the eye, soft palate and pharynx, multiple degenerative neuritis, and, on the mental side, they have had much to do with the psychoses, with the details of which we are specially concerned.

One of the most startling forms of influenza, fortunately rare, was the comatose variety, due to acute intoxication by toxins, characterised by sudden giddiness, or apoplectic, or epileptic seizures, followed by hours of unconsciousness, and attended with high fever, which indeed served as a diagnostic. In children, the attack sometimes began with epileptiform attacks and stupor, going on into a chronic form with seizures at long or short intervals, all, however, ending in recovery.

Dr. Lewis C. Bruce, in his recent book (*Clinical Psychiatry*, 1906, p. 210), says that, in all insanities in which there is evidence of toxæmia of bacterial origin, there is always a marked hyperleucocytosis at some period of the disease. In patients, who recover, there is evidence that the leucocyte-producing tissues are active, and especially the tissues which produce the polymorphonuclear cells. In patients, who do not recover, there is evidence that the power of producing polymorphs is impaired, or exhausted, and there is no exception to the rule that chronic cases of insanity of this class have a deficient percentage of polymorphs. There is also evidence of the presence of specific agglutinines in the serum of patients suffering from toxæmic insanities. In the *Lancet* (1892), Dr. Althaus, writing on this subject, quotes some Danish cases showing intense hyperæmia of the pia mater and the brain, especially in the basal arteries, which resembled thick cords; but, in opposition to these, Goodall quotes instances in which there was neither pachy- nor lepto-meningitis, no atheroma of the arteries, and no congestion. Dr. Hädeke, of Munich, described a case of meningitis and epidural abscess with proof of the presence of Pfeiffer's bacillus, and Cantani found it in the brain substance and in the ventricles in the experimental infection of rabbits.

In the majority of cases, the commencement of a psychosis, or mental affection, is directly after the end of the influenzal attack, less frequently after one or two weeks. The most common form is a melancholia, more rarely it is of a maniacal or excited character, especially in children, and some cases have been reported of acute paranoia, or acute delusional insanity, and even of general paralysis.

Bidou describes the influenzal psychoses as of three kinds, (1) depression, (2) maniacal excitement, (3) idiopathic psychoses coming out on the influenzal advent. These last forms have been already alluded to, and as to the others they may be taken as representing what is generally seen, identical with what we notice in the delirium of convalescence, in pneumonia, in the exanthems, and in the puerperal state. Three things are to be kept in view: first, that, in most forms of the disease, the prognosis is hopeful; secondly, that the tendency to suicidal acts should never be lost sight of, and thirdly, the probability of relapse.

That influenza, even in the most favourable cases, is usually accompanied with, or followed by, a feeling of depression is recognised, and in some (but what percentage cannot be stated) instances this depression becomes a true melancholia, often accompanied by suicidal attempts. The distinction between the common form of simple depression and that of melancholia is well-marked, and should be carefully attended to.

In his recent work on melancholia, Dr. R. Masselon refers to what he calls simple depression and moral grief or pain with anxiety, which he defines as a complex sentiment founded on depression, but with the addition of periodic crises of anxiety. This psychological complex, which is otherwise called acute mental pain, or anxiety, or anguish, comprises such affective or emotional states as painful sentiment, worry about indefinite physical ailments, doubts about the future, feeling of impotence and of incapacity for action, profound want of confidence in the conduction of affairs, etc. An analysis ot this condition by M. Séglas explains it by the presence of two factors, one being the alterations of the cœnæsthesia, or the feeling of common sensation, and the other the arrest of the psychical processes. And there is no doubt about the correctness of Séglas' theory. When we recollect the gastric and pulmonary disturbances which more or less accompany all forms of influenza, the general or limited neuritis, the intense headache, and other signs of lesion of the normal processes, which go to form the whole bundle of influences which constitute the "content" of the common sensation of well- or ill-being, it would be surprising, if there were not some delay or obstruction in the completeness of the psychical flow in the central nervous system, a condition which, according to the latest theories on the subject, is the basis of depression or melancholia, and, in its more developed form, is the cause of that change in the ego of the individual, which declares itself by delusions of persecution, of unfitness for life, of change of identity, and which may even result in acts of violence towards self or others.

What actually determines the impulse to suicidal acts, so common in the depression attending or following influenza, is difficult to say, unless we agree with Masselon, and put it down to "loss of the instinct of self-preservation." Whether such an instinct exists as an entity is more than doubtful. It is true that, normally, all the forces of our organic life are conservative in their direction, and that if any impairment or failure of their action ensues, as the result of a morbid process, there must *pro tanto* be a lessening of this conservative tendency, and so the melancholic, feeling the loss of his powers of resistance, that he no longer needs anything, that his intellectual activity is diminished, loses the most important factor in true complex voluntary action, namely, a correct emotional state, and this induced, imperfect condition of his inhibitory power means that he becomes reduced to a state of simple voluntary action, otherwise called impulse. Some of the patients, who suffer from influenzal melancholia, belong to the "persecuted" class, they think themselves tormented by their enemies, that they are going to fall victims to a plot, and that they are going to be killed, but, instead of giving in to the ideas, they put all their energy into facing the situation, and, instead of injuring themselves, they do what they can to counteract the influence of those whom they imagine to be threatening them. I believe that in these cases the general cœnæsthesia is less affected than in those cases of melancholia, which are especially denoted by depression and acute feeling of anxiety, and that what Hoffling calls the "fundamental tone," produced by the normal course of the organic life, the composition of the blood, the functions of the glandular organs, and the tension or relaxation of the capillary vessels and of the involuntary muscles, is less altered. That there is, in suicidal patients, a profound change in the sensory and perceptive organs is abundantly proved by the fact that the act itself is, as a rule, unattended with pain, and I recently had under notice a case in which the patient was so astonished and puzzled, because she, neither at the time nor afterwards, felt any pain from the injuries caused by a very determined suicidal attempt, that she actually made a second one in order to "see whether she had recovered her sensibility."

Pain is then both a protective feeling and a noteworthy sign to the outsider, and I would rather trust a patient with normal organic sensations, though greatly deluded, than I would one, in whom, with an apparently slight delusional state, there was extensive emotional or affective change. I have ventured to consider rather fully this symptom of the chief form of the influenza psychosis, because it has been my experience that the excess of alteration of the affective tone, and the comparative small prominence of the more excited symptoms render the patient more liable to have his really dangerous condition overlooked, and that, just when he needs care and supervision most, he is apt, from the negative external signs, to run risks to which he ought not to be exposed.

Just as in the ordinary course of an influenzal attack relapses are the rule, so on the mental side must relapse be expected. In severe cases of depression, this tendency must be ever borne in mind, especially with the view of protection from recurrent suicidal attacks. The simple depression, which is so common in ordinary attacks of influenza, may last a long time—more than a year—but it is not usual for the pronounced insanity to last so long as that, and most of the cures occur after a few weeks' or a few months' treatment, though I know of cases which have not recovered after years of detention and treatment. After insisting upon the importance of recognising the two prominent features of suicidal impulse and the tendency to relapse, there is little or no difference in the symptoms of influenzal melancholia, and other forms produced by stress or physical causes generally. Thus, the condition may remain throughout a simple melancholia, or it may be accompanied with delusions of the usual type, viz., of fear, or of persecution, or of self-reproach, or it may pass into a maniacal stage (though this is not common), or it may descend gradually into a condition of dementia, from which there is no recovery. Of all forms of insanity, melancholia is the chief type assumed in the influenza variety. In the cases received at the Banstead Asylum during the years 1890–1905, this was invariably the form, but at St. Luke's Hospital, during the years 1890–1900, out of 25 cases reported, two-thirds were of the melancholia type, and nearly one-third of the maniacal variety, whilst one patient suffered from primary stupor. It is difficult to account for this discrepancy, unless it is explained by the fact that, whilst the cases at St. Luke's numbered females in excess, and that the form of mania was, to a great extent, confined to them, at Banstead the male patients were in excess, and both these and the women were drawn from a different social grade. Hysterical neurasthenia being a frequent result of influenza, it is more likely that women developing an insanity would take on the maniacal, or excited form, but even so, the frequency of the depressed form of the psychosis is by far the most common.

In the majority of cases the commencement of a psychosis is directly after the end of the influenzal attack, less frequently after one or two weeks, but, in rare instances, there is a prodromal stage, lasting one or more days, of acute febrile dementia of the melancholic type. This precedes the development of the influenza, and is not necessarily associated with mental symptoms after the influenza proper has run its course.

Reference has already been made to previous neurasthenic or degenerated states, as predisposing to post-influenzal psychoses, and it may be here mentioned that alcoholics often have attacks of delirium tremens as a result of the influenza poison, whilst children may develop epileptiform attacks and stupor, this form of epilepsy becoming chronic with seizures at long or short intervals, a sort of toxic tetany, ending, however, in recovery. The experience of Bidou, that maniacal excitement is one of the forms of psychosis, is confirmed by what has been stated above, but the liability to this form is much less than in the melancholic kind, and, when it does occur, it is chiefly in women or children. The maniacal attack is characterised by sudden onset, and is most likely attributable to the invasion of the Pfeiffer bacillus. It may be attended with delusions, hallucinations, and violence, and there is nothing particular to distinguish it from the symptom-complex of maniacal attacks due to other causes, but the condition is one generally recovered from, and is of short duration, for which reasons, during an epidemic of influenza, the sudden onset of acute head symptoms accompanied with a high temperature should be prognosed with great care, and the questions of placing the patient under certificates, and of sending him to a hospital for the insane, should be deferred as long as possible, having regard to the rapidity with which these symptoms subside, leaving the true nature of the disease to declare itself. Bidou talks of "idiopathic psychoses" or forms of mental affection of different kinds not characterised by the usual depressed or excited symptoms, lasting some weeks or months, but of favourable prognosis. Such states may be seen in the form of loss of memory, of power of attention and concentration, in neuralgias, or nuclear paralysis, loss of the sense of smell or taste, conditions of doubt and perplexity, known as aboulia and folie de doute, but recovery generally results after tonic treatment, and permanent paralyses, followed by contractions, are never seen. Some writers have described cases of acute paranoia, and even of general paralysis, as resulting effects of the influenzal poison, but such forms are exceedingly rare, and some doubt must be attached as to their real connection with the disease in question. Insidious, however, as are the forms in which influenza appears, it is always necessary, especially during an epidemic, to be prepared for anomalous demonstrations of mental peculiarities, giving ourselves time for the development of their true nature, and recognising that it may not be possible, even at the last, to assign the correct relationship which they hold with the infective process. At times, the melancholia is attended with muscular demonstrations, such as restlessness, rubbing the hands, resistance to attempts to move from place to place, or to adjust the dress, constant picking at the clothing, etc., but here we notice that the muscular action is confined to movements of small radius, very different from the sweeping curves and larger radius of the body and the extremities, seen in the maniacal form. It is probably from want of care in discriminating these two forms of impairment of muscular groupings, that confusion has arisen in classifying the melancholic or the maniacal varieties.

The prognosis, in cases of influenza psychoses, is good. As a rule, the subjects, whether melancholic, maniacal or stuporous, eventually and quickly recover; but a certain number do not recover, and these go on to dementia, or remain in a more or less excited chronic condition.

Dr. Rawes, the medical superintendent of St. Luke's Hospital, has kindly given me the particulars of 25 cases, 18 females and 7 males, of insanity traceable to influenza as the cause, between the years of 1890 and 1900, and the conclusions he draws are as follows :—

1. Males and females are equally liable (they admit into St. Luke's about 3 females to one male).

2. Melancholia is twice as frequent as all other forms of insanity put together.

3. Insanity may come on during an attack of influenza, or at any time up to 18 months afterwards.

4. Of the 25 cases, 8 recovered and 3 were much relieved. If these "relieved" cases are included, the recovery rate would be 44 per cent., a rather unfavourable result, due probably to the severe nature of the admitted cases. Of the 25, 8 remained not improved and there were six deaths, the causes being

pneumonia, heart diseases, and exhaustion from a very broken-down state on admission.

The tendency to relapse was especially noted. The above figures closely correspond with those kindly given me from Banstead by Dr. Johnston Jones, viz., out of 38 cases, 18 recovered, 9 died, and 10 remained "not improved," giving a percentage of 47 per cent. of recoveries and 25 per cent. of deaths. The attacks were about equally distributed between the male and the female admissions and the tendency to relapse was again noted.

The difference between the form of the disease, as noted at St. Luke's and at Banstead, is probably more nominal than real, because authorities often vary as to whether they place some cases of very restless insanity under the head of melancholia, or that of mania. However this may be, there is no doubt of the great preponderance of melancholia in those who suffer mentally in this complaint; and it does seem as if the maniacal or excited form (which is of course a more advanced type of degeneration than the melancholic), is the more unfavourable as regards prognosis.

In the *Journal of Mental Science* for 1901, Dr. Rorie published the details of 68 cases of post-influenzal insanity in the Cumberland and Westmoreland Asylum, and his conclusions were that the psychosis may follow any time after the attack, that it occurred usually in persons with hereditary alcoholic or other predisposition, that it was chiefly of the melancholic and suicidal form, and that the state of mental confusion and stupidity were suggestive of auto-intoxication. Here also the cases were divided, equally between the males and females, and the percentage on the total admissions was 6—7, the average age on attack being 46 years.

The forms of insanity were :—

34 males -	26 melancholia.
	3 acute mania.
	4 mania.
	1 general paralysis.
34 females	20 melancholia.
	4 acute mania.
	8 mania.
	1 senile mania.
	1 dementia.

Dr. Macphail published 20 cases of post-influenzal insanity at the Derby Asylum between the years 1890–1895, the results being about the same as stated above. In none of these accounts is there any record of blood examination for micro-organisms.

Although there seems to be no doubt about the existence of the bacillus in primary meningitis, yet I cannot find that it has been ever discovered in post influenzal insanity. Moreover, as the antitoxins have not hitherto been obtained, the serum treatment may be disregarded, or reserved for those cases of the comatose variety of nervous symptoms, such as sudden giddiness, or apoplectic, or epileptic seizures, followed by hours of unconsciousness, cases which are doubtless due to acute intoxication by influenza toxins, or a true suppurative cerebro-spinal meningitis, such as described by Pfuhl in 1892, where Pfeiffer's bacillus, strepto- and staphylococci were demonstrated.

The treatment must be directed to the exercise of great caution in preventing the patient from doing himself any harm, and to the management of such symptoms as excitability in an expectant manner, avoiding sedatives and narcotics as much as possible, and, keeping in view the fact that, if the mental depression or the excitement does not soon improve, it may be necessary and advisable to place the person under restraint. I have never met with, nor have I seen reports of cases, in which the injection of antitoxins has been resorted to, but the possibility of their value should be held in view, especially, as stated above, in those instances of early delirium, where it is probable that an acute invasion of the bacilli has occurred. Measures directed to the maintenance of the general health by regulated and sufficient diet, and the insistence of absolute rest, are most important, and administration of the glycerophosphates in convalescence may be strongly recommended. Insomnia is often a troublesome symptom, but the temptation to administer sedatives should be strenuously resisted, except in particular instances. I have found that, when it may be properly undertaken, motor-car exercise is most beneficial for procuring sleep. The tendency to relapse must be regarded and precautionary measures for safety not relaxed until the patient has been thoroughly restored to health. I recently saw a lady, who has had several attacks of influenza with a short but very decided condition of restless melancholia following each attack as a post-influenzal complication. The possibility of this successive affection should be always looked out for in cases in which it has once occurred. In the particular instance quoted, the melancholia was of a lower type in the later of the recurrent attacks, and necessitated her temporary confinement under certificates.

In briefly reviewing our knowledge of these post-influenzal psychoses, it may fairly be said that our present position is a strong one. In recognising that the insanity is of a toxic nature, due either to the direct action of the bacilli, or of the toxins, we have proceeded far in the direction of a rational treatment, and, it is to be hoped, that since the connection between the two has been established more detailed examinations of the blood and fluids may, in the future, be made, especially in the institutions to which severe mental cases have to be sent. Upon all who are concerned with the treatment of influenza, the obligation rests to remember that in proportion as the patient is neurotic or degenerate, or has a bad family mental history, so is he more likely to suffer from a post-influenzal psychosis, which, in a small percentage, may end fatally, and which is always a matter of solicitude, because of the tendency to relapse, and of the frequency of suicidal impulse.

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OCULAR MANIFESTATIONS ACCOMPANYING INFLUENZA.

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ALTHOUGH influenza may now be considered to be quite a common disease, which occurs in both epidemic and sporadic form, the ocular complications, which accompany or follow it, are decidedly rare. Many of those ocular affections, which have been described as resulting from influenza, are certainly due to other causes, such as syphilis, or auto-intoxication, which follows the absorption of toxins from the alimentary canal, and the influenza, which occurs, is simply the determining factor by means of which the resistance of the body to the invasion of micro-organisms and toxic bodies is lowered.

The Conjunctiva.—An attack of influenza is often ushered in by an acute catarrhal conjunctivitis, which is accompanied by other congestive phenomena, such as hyperæmia of the mucous membrane of the nose and the frontal and anterior ethmoidal air sinuses. The palpebral and ocular conjunctiva may both be involved in the inflammatory process, which, in some cases, spreads to the episcleral tissue, thus constituting a true episcleritis. These inflammatory conditions are often accompanied by photophobia and lachrymation, and referred pain along the supra-orbital, supra-trochlear, lachrymal, and infra-orbital nerves. Sometimes the patient experiences pain at the back of the orbits. Sneezing may accompany this acute coryza, and sometimes profuse epistaxis takes place.

The Cornea.—The superficial layers of the cornea may be involved in the condition, known as herpes febrilis corneæ, in which there are herpetic vesicles upon the corneæ, the upper lids, and along the forehead in the position of the supra-orbital and supra-trochlear nerves, associated with supra-orbital neuralgia. When the superficial nerve plexus in the cornea, which lies under the anterior homogeneous layer of Bowman, is involved in the inflammatory process, there is usually reflex blepharospasm and a watery discharge from the nostrils.

The Uveal Tract.—The iris, ciliary body, and choroid are rarely affected in influenza. In the congestive stage of the disease, the iris may be somewhat congested, and the pupil therefore contracted, but there are, as a rule, no posterior synechiæ formed, indicating therefore the absence of any plastic iritis. Adler and Fuchs have reported a case of iritis, accompanied by hypopyon and purulent hyalitis; tenonitis followed, and the pus perforated the anterior portion of Tenon's capsule, and so escaped. Glaucoma has sometimes been observed as a sequence of influenza.

The Lens.—Influenza has no direct influence in the production of cataract, but there is no doubt that, if a senile cataract is present, an attack of influenza hastens the maturing of it.

The Optic Nerve.—Bilateral papillitis, during an attack of influenza, and persisting after the influenza has subsided, sometimes occurs, occasionally the papillitis is unilateral. Cases of retro-bulbar optic neuritis have been reported, in which there has been no previous history of excessive tobacco smoking, or chewing, or of the abuse of alcohol. Weeks, who has reported a case, states that the scotoma, which he has observed, is not identical with that which is usually present in cases of amblyopia, due to tobacco and alcohol. Instances of optic neuritis, in which the nerve and its sheath are involved, and which have been followed by consecutive optic nerve atrophy, and therefore blindness, have been reported, although they are fortunately rare. Optic neuritis, which is probably a descending neuritis, may, however, develop as a complication of basal meningitis and intracranial abscess, and may occur during an attack of influenza. The latter condition is usually due to otitis media. It must be remembered, however, that optic neuritis is not nearly so frequently seen with a brain abscess as it is with tumour of the cerebellum or cerebrum.

The Ciliary Muscle.—The muscle of accommodation is frequently affected, and patients find difficulty in reading, threading their needles, and doing their stitching, especially by artificial light. This condition is exactly what would be expected to arise as the result of a specific infectious disease, which leaves the neuro-muscular system in an asthenic state. This weakness of the ciliary muscles is much more likely to arise in young people, who have latent hypermetropia, or hypermetropic astigmatism. The chief symptoms of this asthenopia are the occurrence of frontal headache, and red congested eyes and eyelids after reading, or doing any work requiring close observation, and, after a time, the development of follicular conjunctivitis, or of chronic ciliary blepharitis. In even the simplest cases, it may be necessary to order spherical lenses for a time; as the general tone of the body recovers, the symptoms may disappear. On the other hand, those who are nearing the age of presbyopia, and especially those who have been accustomed to use their eyes a great deal for near work, now find that the use of lenses is essential for the performance of their near work with any degree of comfort. Fuchs states that cases of complete paralysis of accommodation, which were analogous in their behaviour to post-diphtheritic paralyses, occurred fairly frequently during the last epidemic. These paralyses were due to peripheral neuritis of the fibres of the third cranial nerve which supply the ciliary muscle. Both eyes were usually affected; as a rule, the sphincter iridis is not involved.

The Extrinsic Muscles of the Eye-ball.—In the early stages of influenza, patients often complain of the pain, which they experience when attempting to move their eyes, and which is no doubt due to pain in the muscles themselves. In these circumstances, patients often prefer to keep their eyelids closed, and so to avoid undue muscular fatigue. From time to time, cases of paralysis of the muscles supplied by the third, fourth, and sixth cranial nerves, have been reported. In some cases, the condition is one of peripheral neuritis, but occasionally it is of central origin. Dr. Goodhart states that, of the external group of eye muscles, the external recti appear to be most often affected. Arlt has reported a case of insufficiency of the internal rectus muscle. Ptosis and strabismus with diplopia may occur. Greiff describes a case of paralysis of the right cervical sympathetic following an attack of influenza.

The Orbit.—Exophthalmos, with displacement of the eyeball downwards and slightly outwards, may occur in those cases of purulent sinusitis of the frontal and anterior ethmoidal air sinuses, which are due to infection through the nose, following an attack of influenza. One of the earliest and chief symptoms of this inflammation, with sinus suppuration, is a violent supra-orbital neuralgia. It should be borne in mind that these abscesses may appear several weeks after the apparent termination of the attack of influenza.

THE AURAL COMPLICATIONS OF INFLUENZA.

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INFLUENZAL infection may attack the *middle* or *internal* ear.

The middle ear complications are due to the direct spread of infection along the Eustachian tube from an infected nasopharynx. They may be non-suppurative or suppurative. The non-suppurative or catarrhal may be acute or chronic. Acute catarrh may be limited to the mucous membrane at the mouth of the tube, or it may extend up the tube and reach the middle ear. Occasionally an obstinate chronic middle ear affection can be directly traced to an attack of influenza. These non-suppurative conditions are similar to those seen as a result of an ordinary catarrh, and have nothing distinctive in their course or treatment.

The acute suppurative inflammation, on the other hand, has sometimes typical characteristics. It is of extreme violence; the onset and course are rapid, the pain and constitutional disturbance very great. The membrane quickly becomes hyperæmic and bulging with pus. Sometimes sanguineous blebs form on its surface and even on the meatal wall. Perforation soon occurs, followed by copious discharge, which continues for a few weeks. Recovery takes place in a great number of cases without further trouble. In some, however, there is a rapid extension of infection with great destruction of bone, rendering an operation necessary even within a few days of the onset.

The extension depends, as in all other middle ear infections, to a large extent, on the anatomical condition of the antrum and mastoid process. In many adult bones, the mastoid cells are not present; the outer antral wall is very dense, and a layer of dense bone separates the antrum from the diploetic or dense mastoid mass. In these circumstances, the pus is unable to reach the surface of the mastoid process, and, as the drainage from the antrum into the middle ear is imperfect, the patient's life is in danger owing to the ease with which the pus may perforate, (i) a thin posterior antral wall to the posterior fossa and its contents ; or (ii) the thin roof to the middle fossa. Meningitis, or the formation of a cerebral abscess, may be produced by the infection being carried by the veins and their coverings, which pass through the remains of the petro-squamosal suture from the antrum to the middle fossa, without direct extension of caries.

The symptoms of this antral empyema are : the continuance or fresh onset of pain, worse at night, in the ear and side of the head, and the temperature is usually elevated at night ; but the rise may be slight. The pain may be very acute, but sometimes it is only a dull ache. The discharge continues to be profuse and muco-purulent in character, pulsating strongly as it lies in the meatus. On account of the density of the outer antral wall, tenderness may be very slight, but it can be obtained by deep firm pressure ; for the same reason œdema and pushing out of the auricle are not well marked. On looking into the meatus, after all discharge has been cleared away, there will, as a rule, be noticed a sagging caused by œdema of the posterior-superior deep meatal wall close to the membrane. This sign is of the utmost importance, and indicates the necessity for the immediate opening of the antrum.

In infants and early childhood, the outer antral wall is very thin and very easily perforated by pus, which then forms a large abscess high up behind a down and outpushed auricle.

If mastoid cells are present, being always in communication with the antrum, pus quickly invades them, destroying their partitions, forming a mastoid empyema. The process becomes tender and œdematous, and, if unopened by the surgeon, perforation may occur in several situations, causing an abscess to form outside the point of perforation.

The site of perforation may be :—

Externally, in many situations, forming the ordinary mastoid abscess.

Internally, under the deep fascia of the neck (Bezold's perforation), with perhaps extensive burrowing down the neck, or the pus may pass into the neck through the digastric fossa.

There is occasionally a track of cells passing from the antrum right down to the digastric fossa, wholly internal to the mastoid cells, thus allowing of perforation into the neck, without involving the mastoid process at all.

Bulging of the lateral pharyngeal wall is sometimes produced by the pus which has perforated into the neck.

Anteriorly, into the meatus close to the orifice, and closely resembling a furuncle.

Posteriorly, through the thin bony partition which separates the cells from the lateral sinus, forming an extra-dural abscess in the posterior fossa, and it may here extend further, causing meningitis, thrombosis of the lateral sinus, or cerebellar abscess. The destruction of this partition is exceedingly common in acute mastoid empyema.

At the tip of the process causing an abscess in the neck muscles.

The treatment in the early stage must be prompt, the membrane should be incised under an anæsthetic, and with antiseptic precautions. The incision should pass from well in front, downwards through the inferior segment, and then upwards through the posterior segment right up to the outer attic wall just behind the malleus, the knife being kept as close as possible to the periphery.

A light antiseptic dressing should then be applied and changed three or four times a day, according to the amount of discharge. The ear should be gently syringed with hot boiled boracic acid solution, or sterile salt solution at each change. The patient should remain in bed, and pain may be treated with morphia and hot fomentations applied over the dressing.

A careful watch should be kept for any sign or symptom of extension, and, if they arise, there should not be any delay in operating.

In opening the antrum and mastoid cells in these cases, it is necessary to open the cavities freely by removing their outer walls, and to destroy all bony partitions, always remembering that the lateral sinus is often exposed. If the perforation has taken place through the mastoid process, the outer antral wall need not be wholly removed, but, a free opening should be made from the lower part of the antrum into the mastoid wound. It is unnecessary to touch the middle ear itself, for, so long as the drainage is free, the membrane will heal completely with little or no diminution of hearing.

The large resulting cavity should be *very lightly* packed, and allowed to heal from the bottom, sometimes secondary suture of the skin wound will hasten the healing.

Occasionally it is very difficult, or even impossible, to distinguish between intracranial complication of the middle ear inflammation and symptoms due to systemic influenzal infection. There may be continued hyperpyrexia, or symptoms closely resembling meningitis, thrombosis of the lateral sinus, or even cerebral abscess, co-existent with the ear affection, and sometimes it is only by exploratory operation that the ear as a cause can be excluded.

If a chronic middle ear suppuration is present before an attack of influenza, it is liable to fresh infection, and to rapidly blaze up with acute signs and symptoms of extension.

An acute influenzal inflammation may lead to a chronic condition, due to localised caries of the ossicles, or bony walls, and changes in the living membrane.

The internal ear may be attacked by the extension of suppuration from the middle, in either the acute or chronic stages. During the acute stage the external semicircular canal is more likely to be opened, while in the chronic the neighbourhood of the oval and round windows and the external canal are the most frequent sites. Extension of suppuration to the labyrinth is characterised by giddiness, tinnitus, increased and profound deafness, and vomiting.

Apart from suppuration, there are occasionally seen during an influenzal attack symptoms which point to an acute neuritis of the auditory nerve. The patient complains of giddiness, tinnitus, and deafness, usually in one ear only. Diminished tone conduction with the tuning-fork indicates the nervous nature of the affection. Facial paralysis, or paresis, and loss of taste on the affected side are sometimes noticed, and herpes on the auricle, face, and neck are occasionally associated. The treatment should be : rest in bed, functional rest by plugging the ear, purging with calomel, blistering behind the ear, and the internal administration of bromides in the early stages, and iodide of potassium later. Recovery is the rule.

Patients sometimes complain of a gradual deterioration of hearing in both ears without any other symptom after an attack of influenza, and it will be found that the nervous part is affected. It is supposed that this is due to a chronic auditory neuritis, but it may be central in origin. The treatment is unsatisfactory; rest, change of air, counter-irritation behind the ears, and the administration of iodide of potassium, and occasional course of strychnine, or phosphorus, may be beneficial. Tobacco and alcohol should be entirely cut off.

THE MICRO-ORGANISMS OF INFLUENZA.

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The bacteriology of the epidemic of influenza which occurred in the early nineties, coming as it did in a period of great activity in pathological research, was naturally the subject of careful study, and the discovery of the micro-organism which appeared to cause the disease was first announced by Pfeiffer in 1892, and soon corroborated by numerous observers in all parts of the world. The *Bacillus influenzæ*, or Pfeiffer's bacillus, is now well known though the difficulties attending its cultivation have hitherto prevented the acquisition of such a complete knowledge of its characteristics as is obtainable in the case of most of the other important pathogenic bacteria. It is extremely small, rarely measuring more than $\cdot 5\ \mu$ in length, *i.e.*, it is roughly only one-sixteenth of the diameter of a red corpuscle long. It forms no spores, has no flagella, and is non-motile. It does not stain by Gram's method, and requires the use of rather powerful dyes for its demonstration. When properly stained it often shows a cap of colour at each end with a clear space intervening. This "polar staining" may lead to its being mistaken for a diplococcus if carelessly examined.

The main cultural characters of Pfeiffer's bacillus are these: It is a strict aërobe, requiring the presence of oxygen for its activity; it grows best at the body temperature, and not at all below 27° C. or above 42° C.; and it refuses to grow except on a medium containing hæmoglobin.* The most convenient medium for its cultivation is ordinary agar which has been streaked with a little blood; this may be taken from an ordinary skin-puncture, provided that careful precautions as to asepsis are preserved, and that the medium is inoculated for a day or two before being used, so as to ensure sterility. When grown in this way it forms very minute transparent colonies, which look like drops of dew. Even in the most favourable circumstances it grows feebly and dies out in a few days if it is not frequently transplanted. Involution forms (threads, chains, etc.) are frequently formed in cultures.

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* This is not strictly true, as it may be grown with some difficulty on other media containing certain animal substances (Cantani and others).

A point of considerable theoretical interest has recently attracted some attention. This is the ease with which the influenza bacillus will grow without hæmoglobin, provided that certain other bacteria accompany it. This was first pointed out by Cantani, who succeeded in obtaining cultures when the organism was inoculated on ordinary media along with diphtheria bacilli, staphylococci, and other organisms, and found also that the latter would act in this way even after they had been killed by heat. Luerssen[1] found that the most suitable "nurse" for the influenza bacillus was the *B. prodigiosus*, and added the interesting fact that cultures of this bacillus, killed by being heated to 60° C. retained this action, whereas those heated to 100° C. were inert. Neisser[2] recommends the use of the xerosis bacillus, and succeeded in keeping the influenza bacillus alive for twenty generations when the two were grown symbiotically. In some cases the latter organism was found to be still living when the cultures were twenty days old. There are also some reasons for thinking that killed cultures of streptococci may increase the virulence of the bacillus for animals. If this should prove to be the case it opens up an interesting field for speculation as to the origin of the epidemics of influenza, suggesting that Pfeiffer's bacillus may live, possibly for long periods, as a harmless saprophyte in conjunction with other bacteria, and be suddenly raised in virulence as a result of its entrance into the body in conjunction with the streptococcus, or some other pathogenic bacteria. As we shall see, bacilli which, if not actually identical with Pfeiffer's bacillus, have the closest possible resemblance thereto, are widely distributed, and produce diseases which are constantly present; and it is easy enough to imagine that these might receive, in some such fashion, the slight alteration in their metabolism necessary to transform them into the pathogenic bacillus which occurred in the pandemic in the nineties.

The pathogenicity of the organism for the lower animals is but slight, and the effects following its injection are not similar to influenza in man. Injected into the peritoneal cavity of guinea-pigs in rather large doses it causes acute fatal peritonitis. Animals can be immunised against it, and the immunity is said to depend on the presence of a bacteriolysin, but the common presence of the organism within the leucocytes in the sputum in influenza would suggest that an opsonin plays an important part in this respect. The immunity left in man by an attack of the disease is of short duration.

Several experimenters have produced sera against this infection. In some cases, these were of some value in experimental infections, but I do not know whether they have been tried clinically [3, 4].

The pathology of the disease is not fully known, the rarity of autopsies during the acute stage rendering its investigation difficult. In most cases the lesion is limited to the respiratory mucosa, sometimes the cavity of the nose, and occasionally only the tonsil [5] being inflamed, though usually the disease extends to the bronchioles, or alveoli of the lungs. More rarely other regions, such as the conjunctiva and (in one case at least) the urethral mucosa, are affected primarily.[6] The bacillus can usually be demonstrated with ease in the secretion from these lesions, the simplest method being to spread a thin film, dry, fix, stain deeply with carbol-fuchsin, aiding its action by gentle heat, wash thoroughly in a stream of water, and finally to dry and examine with a high power. In such preparations Pfeiffer's bacillus may often be seen in pure culture; frequently, however, other organisms are present, and there is no doubt that secondary or concurrent infections, though not necessary, play an important part in the pathology of the disease. For example, the pneumonia of influenza may be due to Pfeiffer's bacillus alone, or to this organism in conjunction with the pneumococcus, *M. catarrhalis*, streptococci, or other bacteria. The inflammatory sequelæ of influenza (such as otitis media) are mostly due to these other organisms, especially pneumococci and streptococci.

In some cases the bacillus enters the blood-stream, converting the disease into one of septicæmic type; the frequency with which this occurs is somewhat doubtful. Thus Canon [7] found them in blood-films in all cases of the disease, whereas Klein[8] found them only in six cases out of forty-three, and other observers have not succeeded in finding them at all. Further, Klein failed to cultivate them from the blood in the six cases in which he found them in films, thus indicating that the disease is not of septicæmic type, and that the bacilli which gain access to the blood-stream are rapidly killed.

Horder[9] found living influenza bacilli in the blood in two cases of ulcerative endocarditis, thus proving that living bacilli may circulate in the blood, and become engrafted on distant organs. A case of peritonitis due to this bacillus has also been recorded.

The general and remote symptoms of influenza must doubtless be attributed to the toxin of the bacillus, whether produced locally, in the inflamed mucosa, or liberated by the solution of the bacilli in the blood-stream. It seems highly probable that the latter is an important source of toxin, and one which is not sufficiently recognised. It affords an explanation of the fact that the nervous and other symptoms of influenza are by no means proportionate to the severity of the primary lesion. Thus in the cases in which there are severe general and remote symptoms with very few signs of local respiratory or other disorder it is likely that many bacilli gained access to the blood, were destroyed, and liberated their toxins into the circulation.

The pathology of the gastro-intestinal and other atypical forms of influenza is unknown ; the organism may, for all we know, attack the mucous membrane of the digestive tract, or the symptoms may be due to remote toxic action.

But little is known of the nature of the toxin of Pfeiffer's bacillus. Filtered cultures show no toxicity, and the endotoxin has been investigated by Slatineano,[10] using Besredka's[11] method of dissolving the bacilli in fresh serum and removing the bacilli by centrifugalisation. When injected into the brain of a guinea-pig it kills in small doses, but has little action in the peritoneum.

So far no serious difficulties have arisen. Observations, however, since the earlier years of the epidemic, have shown that the facts are by no means so simple, two disturbing facts being brought to light. These are, first, the existence of bacilli which are indistinguishable from those of Pfeiffer, but which do not cause influenza, and, secondly, the fact that a disease clinically identical with influenza may be caused by organisms other than this bacillus.

One of the first of the influenza-like bacilli to attract attention was that which occurs in abundance in the sputum in whooping-cough, and first observed by Sprengler and by Eppendorf in 1897. This organism has the closest possible resemblance to Pfeiffer's bacillus, the only difference being (according to some only of the observers who have investigated it) that it is slightly larger, a feature that depends on so many factors (especially on the staining method employed), that it is unsafe to attach much importance to it. The two bacteria are identical in their cultural characters, and both show the same symbiotic relations to other organisms. The effects of the inoculation of the whooping-cough bacillus on to the nasal mucosa of man (as described by Davis[12]) is at least closely similar to that of Pfeiffer's bacillus similarly applied, as described by Cantani.[13] And Wollstein[14] has recently shown that the serum of animals immuned against Pfeiffer's bacillus, agglutinates the *B. pertussis* and *vice versâ*, though the serum is more potent against the organism used for the injection. Taking all the facts into consideration we must come to the conclusion that there is no sufficient evidence to show that the organisms are not identical, and that it is a mere begging of the question to distinguish between a "true" and a "false" bacillus of influenza in the case of two bacteria which are indistinguishable, but which come from different sources.

Another important and widely distributed organism closely allied to Pfeiffer's bacillus, and possibly identical therewith (though the evidence is not so strong as in the preceding case), is the bacillus of epidemic conjunctivitis associated with the names of Koch and Weeks, and recognised before the discovery of the influenza bacillus. The bacillus described by Müller in trachoma is also very similar, and at least three other organisms which are identical in morphological and cultural characters have been described. Most writers have fenced with the question, and refused to give a decided opinion as to the identity or non-identity of these bacteria, but Iundell[15] has taken the logical step of asserting that they are all accidental modifications of the same species, and regards conjunctivitis due to the Koch-Weeks bacillus as a form of ocular influenza. This view would at least explain the presence of bacilli which are potentially those of influenza in the absence of an epidemic, though it would naturally leave us to seek the causes, whether symbiotic, atmospheric, telluric, etc., which led to its assuming such remarkable pathogenic properties.

The occasional non-occurrence of Pfeiffer's bacillus in disease clinically identical with influenza has been noticed, in all probability, by most bacteriologists during the last few years, and has been attributed to defective technique, unsuitability of the material examined, or to an incorrect diagnosis of the disease from which the patient is suffering, and such cases have not been recorded. Lately, however, the discrepancy has become very obvious, and observations made in England, France, Germany, and America have shown that in attacks which are clinically typical influenza the bacillus may not be forthcoming. This was first pointed out in this country by Bulloch,[16] and since then Dunn and Gordon[17] have published an account of an "epidemic simulating influenza," the bacteriological aspect of which was studied with much care; the main feature was the absence of Pfeiffer's bacillus, and the frequent presence of *Micrococcus catarrhalis*, to be described subsequently. An even more interesting epidemic was investigated by Besançon and de Jong,[18] who found Pfeiffer's bacillus present in a few instances only, the pneumococcus almost constantly, and occasionally other organisms, such as the pneumo-bacillus, streptococci, pseudo-diphtheria bacilli, etc. In the majority of cases they also found *M. catarrhalis*, or an organism resembling *M. tetragenes.*

Other epidemics with similar bacterial associations have been described, but it is unnecessary to labour the point, as it must be perfectly familiar to all who practise clinical bacteriology at the present day. Pfeiffer's bacillus is still met with but it is comparatively rare, and the most usual organism to find in the sputum or nasal discharge of cases of clinical influenza nowadays is *M. catarrhalis;* after that pneumococci, streptococci, pseudo-diphtheria bacilli, etc.

The *M. catarrhalis* was first described by Seifert in 1896, and was found soon after this by Pfeiffer in conjunction with his bacillus in the lungs in broncho-pneumonia in children. Since then it has gradually become well recognised as a cause of numerous forms of inflammation of the respiratory tract, varying from mild catarrh to severe broncho-pneumonia. It is a diplococcus, often of rather large size, which does not stain by Gram's method, and thus presents some resemblance to the gonococcus and to the meningococcus; when examined in films of pus the resemblance is heightened by the fact that the organisms are frequently intracellular. There s no doubt that confusion has arisen in the past, but with our present knowledge it is not difficult to discriminate between them. *M. catarrhalis* grows at the room temperature, thus distinguishing it from the other two. It forms greyish colonies, which spread somewhat, and does not liquefy gelatin. When cultures are examined the cocci are found to be variable in size and arrangement, diplococci being mixed with short chains, isolated forms, etc.[19]

Thus we see that what is apparently Pfeiffer's bacillus does not always cause influenza, and the influenza (regarded as a clinical entity) is not always caused by Pfeiffer's bacillus. What explanation are we to give of these facts? Two solutions present themselves — (1) that influenza is to be regarded as a specific disease due solely to Pfeiffer's bacillus, and that other disease clinically identical, but due to other organisms, must be called "pseudo-influenza" or some other question-begging name; or, (2) that it is not a pathological entity, but a group of diverse diseases due to various bacteria. There are serious difficulties connected with either explanation.

With regard to the former explanation, it must be pointed out that, although causation is the soundest basis for nomenclature, we are not justified in separating diseases which are clinically identical into different groups unless we can show some good reason for doing so. In the present case the physician would hardly thank us if we told him that a patient in whom he had diagnosed influenza was not suffering from that disease because Pfeiffer's bacillus was absent, but was really ill of a disease identical in symptoms, course, event, sequelæ, and treatment. If we could offer him a potent serum in the "true" cases, the matter would be different, but at present no good would result from attempting to alter the nomenclature which has been accepted for centuries.

Further, if the diseases occurred in well-defined epidemics, which did not blend with one another, there might be some excuse for this view. But this is not the case. The organism, present in the earlier years of this epidemic was Pfeiffer's, and it was usually pure or nearly pure. At a later period, this bacillus and *M. catarrhalis* were frequently found in association in the same patient, and in some of the most recent epidemics *M. catarrhalis* often occurs unmixed with other bacteria, and in the same epidemic some patients may show Pfeiffer's bacilli, some *M. catarrhalis*, others yet other bacteria. It is absurd to term some "true" and some "false" influenza.

We are led to the second possibility, that influenza is not a specific disease, but one which may be due to a variety of bacteria. This does not solve the difficulties, but raises fresh ones. For instance, how can we explain the fact that the toxins of these various organisms produce symptoms which are clinically indistinguishable? And these symptoms are not necessarily those which are caused by the organism in question when not associated with an epidemic of influenza; thus *M. catarrhalis*, in ordinary circumstances, causes mild broncho-pneumonia, which is much less severe than that of influenza.

But a more difficult question arises regarding its epidemicity. We may understand that *one* organism may suddenly become virulent, though we cannot explain how; but it is difficult to imagine how the numerous bacteria which we may find in influenza at the present day can have undergone this rapid change. It is a new feature in epidemic diseases.

The only possible solution that is entirely satisfactory is absolutely hypothetical and unsupported by any evidence. It is that influenza is a specific disease, due to a definite single cause, but that this cause is undiscovered, and perhaps unsuspected; it may be an "invisible" microbe, a protozoan, or some depressing "influence" acting directly on the human constitution, and of a nature as little known as were bacteria when influenza received its name.* We must assume that this factor has some peculiar action on the body, causing definite symptoms, and has in addition the power of temporarily lowering its resistance to certain organisms. If Pfeiffer's bacillus is identical with the other members of the group of hæmophilic bacteria it is a widespread organism, and there is no difficulty in seeing how it might take advantage of this breach in the defences of the body ; and the other organisms are quite commonly met with in the mouth, air, etc.

This view of the pathology of influenza is similar to that generally held with regard to scarlet fever. In the latter disease the cause is unknown, and the local lesions and complications are due to common bacteria acting on tissues of lowered resistance.

* Influenza was formerly attributed to an excess of ozone in the air (Schönbein), to an excess of negative electricity, and to terrestrial magnetism. The first condition, if it occurred, might well injure the respiratory mucosa and allow access to various organisms. Sir Thomas Watson, writing in 1857, alludes to these theories as being more probable than the germ theory, then attracting attention. The chapter is worth reading.

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INFLUENZA FROM A PUBLIC HEALTH STANDPOINT.

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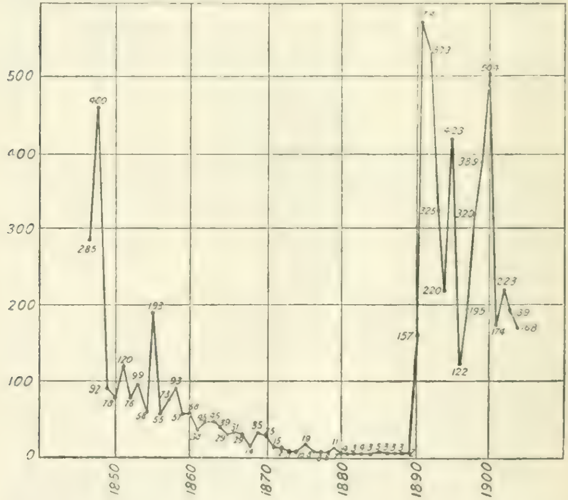
Since influenza is due to a specific germ, introduced into the human body from without, it is, at least in theory, a preventable disease. Whether it is really so depends on the circumstances governing its spread, which will be considered in this paper. Before considering these it may be well to state briefly the extent of its fatal prevalence in this country since statistical returns of causes of death became available.



The accompanying figure gives the annual death-rate per million of population caused by influenza in England and Wales. The highest figure was reached in 1891, when influenza was the registered cause of 574 deaths in every million of the population. This does not represent the total additions to the mortality due to epidemic influenza. In years when influenza is specially prevalent, diseases of the respiratory organs cause an excessive number of deaths. This will be seen by a comparison of the death-rate caused by these diseases in epidemic and non-epidemic years respectively, as shown in the following table :—

TABLE I.

	Non-epidemic Years.			Epidemic Years.		
Death-rate per Million of Population from	1887.	1888.	1889.	1890.	1891.	1892.
Influenza - - -	3	3	2	157	574	533
Bronchitis - -	2,117	2,041	1,957	2,333	2,593	2,266
Pneumonia - -	1,113	1,093	1,022	1,404	1,471	1,250

Influenza is responsible for a large indirect increase of the death-rate, in addition to the heavy mortality directly ascribed to it. Respiratory diseases, as seen in the above table, show a greatly increased death-rate in years in which influenza is epidemic. Were it possible to state statistically the increase of diseases of the nervous and circulatory systems, it is likely that this would be almost equal to the increase in diseases of the respiratory system.

The preceding diagram brings out the point that influenza is not constantly present to a considerable extent in our midst. The history of influenza shows this even more strikingly, but the diagram suffices for our purpose. It will be noted that, after the epidemic which culminated in 1848 and recrudesced in 1855, the death-rate from influenza declined almost to the vanishing point; and, in each of the years from 1881 to 1889 inclusive, it was the recorded cause of only 2 to 5 deaths for every million of population. In the years following 1889 the lowest death-rates from influenza were 122 per million in 1896, and 168 in 1904.

The differences between the curves of epidemic influenza in 1848–55 and in 1890–1904 deserve passing commentary. In the more recent period, three epidemic peaks are visible, and the amount of influenza, constantly present between 1890 and 1904, was much greater than in 1848–55. The statistics for earlier years than 1848 do not throw any light on the difference. In 1843–46, the causes of death in England and Wales were not tabulated, but, in the five years 1838–42, the death-rate from influenza ranged between 53 per million in 1842 and 106 per million in 1841. It is probable that the closer intercommunication between the inhabitants of different parts of England and Wales, which now holds good, is, to a large extent, responsible for the greater magnitude of the recent epidemic of influenza, and for its epidemic recurrence at short intervals of years. At the present time, although influenza is still present to a considerable extent, it appears to be gradually diminishing in virulence, and it may be hoped that, as in the case of epidemic diphtheria, which has followed a somewhat similar course, it will in a few years again become an insignificant quantity.

Importation of Infection.—Whatever view may be taken as to the relation between true influenza and the more generally prevalent "influenza colds" of the community, it is certain that the true, or Russian, influenza was freshly introduced into this country in 1889 90. No one can doubt this who has the facts fresh in his mind, or who refers to Dr. Parsons' admirable report on the subject to the Local Government Board. I do not propose to argue this point; but to start with the assumptions to which the evidence irresistibly points: (*a*) that influenza does not travel faster than human communications; and (*b*) that it is spread exclusively by direct or indirect infection. That this course is justified may be gathered from the following quotation. The late Sir R. (then Dr.) Thorne Thorne states, in his introduction to Dr. Parsons second report (May 1893): – "The evidence which is now forthcoming tending to show that the disease has followed the lines of human intercourse, so far outweighs any that has been put forward in favour of its being due to 'atmospheric causes,' as to suggest that, where these have been accepted as accounting for outbreaks, all the circumstances of the local prevalences in question have not been forthcoming." This, at the time it was written, represented a great advance of medical opinion. Thus Dr. Symes Thompson (in his *Influenza: an*

Historical Survey, p. vii, 1890,) wrote: "Unlike cholera, it (influenza) often outstrips in its course the speed of human intercourse." This appears to be a repetition of a statement on p. x of the *Annals of Influenza*, by Dr. Theophilus Thompson, 1852, and may not, so far as I know, represent the views of the first named. In the epidemic of influenza in 1836-37, a joint inquiry was made by the Provincial Medical Association of England, and the medical answers to inquiries as to the infectivity of influenza were "of an almost uniform tenour, the opinion of nearly all those who had the most extensive opportunities of investigating the disease, and the best means of arriving at a definite conclusion, being that there is no proof of the existence of any contagious principle, by which it was propagated from one individual to another." With more exact inquiry and investigation, it has become clear that infection explains, as completely as can be expected, the spread of the disease; though it leaves us in a condition of uncertainty as to the reason why at long intervals influenza becomes widely epidemic and pandemic.

Can the Spread of Infection be prevented?—If influenza spreads by means of infection, can this spread be prevented? The answer must be yes, in view of actual experiences of such prevention. The details given in Dr. Parsons' reports and in other documents show that in isolated communities, as in certain institutions, in lighthouses, and so on, influenza is absent, when severely prevalent in neighbouring districts.

In institutions for the aged, such as workhouses, this means of prevention is particularly important, and might with advantage be more systematically practised; though, in order to be successful, an accuracy and completeness of supervision and a rigid enforcement of regulations liable to break down at many points are required. Thus the occupants of the Brighton Sanatorium (Isolation Hospital), being strictly isolated in every respect, escaped during the first two outbreaks of influenza in the recent epidemic period, and, in the third epidemic, escaped until a servant, who had been absent on leave for a few days, returned to the Sanatorium and almost immediately developed influenza. Her sister caught the disease from her, but, these two patients having been isolated, no further spread of infection occurred in the institution.

In the general population, the above question cannot be answered with more than a very partial affirmative. There are numerous difficulties in preventing the spread of infection :—

(1) The period of incubation is very short, and thus the multiplication of infection is much more rapid than in diseases like measles and small-pox, in which 12 to 14 days elapse between infection and illness.

(2) The disease is infectious in the earliest stage before its nature is recognised.

(3) The milder varieties of the disease, in which the patient is not disabled from work, are probably as infectious as the more severe attacks; possibly more so in view of the greater freedom of the first class of patients.

(4) The diagnosis of influenza is often difficult. It has frequently been confused with ordinary catarrhal attacks, with inflammatory conditions, with typhoid fever, and with many other diseases.

For these and other reasons, it does not appear likely that any isolation of infectious patients, on a sufficiently large scale to influence the result, can ever be successfully enforced, even if every patient known to be suffering from influenza, were willing to submit to such isolation.

In view of similar considerations, it appears highly improbable that any good would result from the proposal, occasionally made, that influenza should be added to the schedule of diseases which it is obligatory to notify to the medical officer of health. Such notification would obviously be extremely incomplete, and incomplete notification in the case of a disease with a very short incubation-period, a very early infectivity, very difficult of diagnosis, and assuming many clinical forms, offers few prospects of success.

Disinfection of invaded houses does not appear to be required. There is little, if any, evidence that the infection hangs about premises for more than a few days. In this respect influenza appears to resemble measles, in which also the contagion is very short-lived. Domestic cleanliness appears to be what is specially required, apart from the disinfection and proper disposal of expectoration, and the prevention of spread of infective material by pocket handkerchiefs, etc.

The above brief review does not appear to be very hopeful from the standpoint of public health. We have to recognise that we do not, at present, know why at long intervals—over forty years in this country in the last interval—influenza becomes widely epidemic. Assuming a present epidemic, the only hope of preventing the spread of infection is by the careful isolation of infected persons. The duration of isolation which is desirable is doubtful ; but it is likely that, if every influenzal patient would consent to keep indoors for ten days, a large mass of infection would be prevented. The public are becoming familiar with the idea of the infectivity of influenza, and we may hope that the public health conscience of the community will in time be braced up to the point of self-sacrifice involved in the non-compulsory isolation which this case demands. This is the more likely to occur, in view of the consideration that fatal result in influenza is very apt to be the consequence of premature return to work.

I have not dwelt upon the fact that one attack of influenza appears to afford no protection against further attacks. In this respect influenza somewhat resembles erysipelas, and the exceptional cases of scarlet fever and diphtheria, in which relapses occur, and in which infectivity persists for many months. Such cases of influenza may, and probably do, explain the recurrence of outbreaks of that disease, when there has been no known recent exposure to infection.

BY-PATHS OF MEDICAL HISTORY.

INFLUENZA IN THE PAST.

FOR sixteen years we have had influenza more or less always with us; and familiarity, instead of breeding contempt, has engendered an increasing fear of the disease. When it came upon us in the last days of 1889, there were few old enough to remember a previous visitation, and it was generally looked upon as a new thing. Till our own day, when its long stay has given us the opportunity of knowing it too well, it appeared at intervals sufficiently long to make it new and strange to the generation upon which it fell like the shafts of Apollo on the Danaans. There are records of its prevalence at different times extending back to the twelfth century, and it is most likely that it has afflicted the human race intermittently since the ape of "the days that were earlier" got a thumb to his fist, and "became man—and a Positivist." The history of all known epidemics has been written by Hirsch in his *Handbook of Geographical and Historical Pathology* (vol. I., Chap. I.); while the chronicles of its descents upon our own shores, from the sixteenth to the nineteenth century, were collected by Dr. Theophilus Thompson, the father of Dr. Symes Thompson who recently passed away, into a volume entitled *Annals of Influenza or Epidemic Catarrhal Fever in Great Britain*, which was published by the Sydenham Society. Dr. Charles Creighton in his classic *History of Epidemics* also gives a brief account of the visitations of influenza from which this country has at different times suffered. From these various sources, supplemented here and there with details gathered elsewhere, the following brief review has been compiled.

In 1173, a disease presenting some of the characteristic features of influenza as we now know it, overran Italy, Germany and Great Britain. The chronicle of Melrose Abbey for that year mentions "a certain evil and unheard of cough (*tussis quædam mala et inaudita*)" which attacked nearly every one and caused many deaths. A disease, the characters of which correspond with those of influenza, is alluded to in early Gaelic manuscripts under the name of "Creatan," which is probably connected with "Creat," the chest. In the *Report* of the Medical Department of the Irish Local Government Board for 1890, influenza is said to be the same as an epidemic disease which under the name of "Slaodan," or prostration, prevailed in Ireland in 1326 or 1328; in the *Annals of Clonmacnoise* the same epidemic is termed "murre." The same term occurs in a chronicle kept by a nameless monk of St. Albans. Under 1427, he makes the following record: "In the beginning of October a certain rheumy infirmity (*quædam infirmitas rheumatiga*) which is called 'mure' invaded the whole people, and so infected old and young that it carried a great number to the grave." Dr. Creighton thinks that "murrain" or *morena*, which occurs in Latin chronicles, is probably the modern survival of "mure" or "murre."

The first visitation of which we have a full description occurred in 1510. In that year a pandemic wave of influenza swept over Europe. The following account of it is given by Thomas Short in his General Chronological History of the Air, Weather, Seasons, Meteors, etc. (London, 1749):—

"The disease called *Coccoluche* or *Coccolucio* (because the sick wore a cap or covering close all over their heads) came from the island *Melite*, in *Africa*, into Sicily; so into *Spain* and *Italy*, from that over the *Alps* into *Portugal*, *Hungary*, and a great part of *Germany*, even to the *Baltic Sea*; every month shifting its situation with the wind from east to west, so into *France*, *Britain*, etc., *Valeriola*, *Pechlin*, etc. It attacked at once, and raged all over Europe, not missing a family and scarce a person. A grievous pain of the head, heaviness, difficult of breathing, hoarseness, loss of strength and appetite, restlessness, watchings, from a terrible taring cough. Presently succeeded a chilness, and so a violent cough, that many were in danger of suffocation. The first days it was without spitting; but about the seventh or eighth day, much viscid phlegm was spit up. Others (though fewer) spit only water and froth. When they began to spit, cough and shortness of breath were easier. None died, except some children. In some it went off with a looseness; in others by sweating. Bleeding and purging did hurt. Bole Armoniac was chiefly useful, with only lintus's, pectoral troches and decoctions. Where blood was let, the disease proved malignant and pestilential, being attended with a violent, cruel, and unheard of malignity, and made bad work."

In Paris, the complaint was so universal that the ordinary greeting among friends was "*N'en as-tu pas goûté un morcelet?*"

In France, several times in the sixteenth century the sittings of law courts had to be suspended, and preaching perforce ceased in the churches on account of the incessant coughing of the congregation.

Dr. Creighton suggests that the "sweating" by which, according to Short, the disease "went off," is referred to by Erasmus in a letter written from Cambridge in 1511, where he tells his correspondent that he is still weak *a sudore illo*. He may, however, mean the sweating sickness, which seems sometimes to have prevailed in one country, while influenza raged in another; it is not unlikely, therefore, that in those days, when clinical observation was in its infancy, there may have been some confusion between the diseases. In the early years of the sixteenth century, England was devastated by epidemics, which were indiscriminately called the "plague." How easy a prey this country, which now boasts itself the leader in sanitary progress, then offered to infectious disease may be gathered from the account of the manner of living of our forefathers given by Erasmus to Cardinal Wolsey's physician. Although it is well known, it may appropriately find a place here:—

"I often grieve and wonder how it happens, that Britain has now, for so many years, been afflicted with a continual plague, and chiefly with the sweating-sickness, which is a malady that seems almost peculiar to the country. We have read of a state being delivered from a long-continual pestilence by changing the style of building, upon the advice of a philosopher. If I am not deceived, England may be freed in a similar manner. In the first place, the English have no regard to what quarter of the heavens their windows or doors are turned; in the next, their sitting-rooms are generally so constructed, as to be incapable of being ventilated, which is a thing that Galen particularly recommends. Furthermore, a great part of the wall is made transparent by glass plates (or squares), which admit the light, but exclude the wind; and yet, through the small crevices, they admit the air to be strained, which becomes somewhat more pestilent by staying there a long time. The streets, too, are generally covered with clay and rushes, which are so seldom renewed, that the covering sometimes remains twenty years, concealing beneath a mass of all descriptions of filth, not fit to mention. Hence, upon a change in the atmosphere, a certain vapour is exhaled, in my opinion, not at all wholesome for the human body. Added to this, England is not only surrounded by the sea on every side, but it is also, in many places, marshy, and intersected by salt streams, to say nothing at present of the salt food, of which the common people are amazingly fond. It is my firm opinion that the island would become much more wholesome if the spreading of rushes on the ground were not used, and if the chambers were so built as to be exposed to the heavens on two or three sides, the windows of glass being so made as to open altogether, and close in the same way, and to shut so as not to admit noxious winds through the crevices. Since, as it is sometimes wholesome to admit the air, so it is sometimes as much so to keep it out. The common people laugh if a person complain of the cloudy sky. If, even twenty years ago, I had entered into a chamber which had been uninhabited for some months, I was immediately seized with a fever. It would contribute to this object (to render the island more healthy) if more sparing diet could be more generally recommended, and a more moderate use of salt provisions; and if certain public officers were commissioned to keep the roads more free from nuisances. Those parts, too, should be looked to more particularly which are in the neighbourhood of a town. You will laugh at my having time to trouble myself about these matters. I love the country which has for so long a time given me an hospitable abode, and in it, should circumstances allow, I would willingly spend what remains of life. I have no doubt, from your character for wisdom, that you know these matters better than myself: I resolved, however, to mention them to you, that you may, if my opinion coincides with yours, recommend these hints to the notice of the great. For, in former days, kings were wont to interest themselves in such things," etc., etc.

From this letter it might be claimed for Erasmus that he was an early apostle of the gospel of the open window.

In 1557, "a very strong cold North wind" was followed by "many catarrhs quickly followed by a most severe cough, pain of the side, difficulty of breathing, and a fever." On the third day the patients expectorated freely. "The sixth, seventh, or, at the farthest, the eighth day, all who had that pain of the side died; but such as were blooded the first or second day recovered on the fourth or fifth; but bleeding on the last two days did no service." It was noted that, in the 1510 epidemic, bleeding did hurt, and in 1557, though we are told that bleeding saved life in England, it appears to have worked fearful havoc in Spain. At Mantua Carpentaria, three miles from Madrid, bleeding or purging was so dangerous that "in the small town 2,000 were let blood of and all died."

It may safely be assumed that, between the great pandemic waves of influenza, there were small local outbreaks which found no sacred bard to record them. Of one of these at Edinburgh, we get a glimpse in a letter written in November 1562 by Randolph, the English Resident at the Scottish Court, to Cecil. He says:—

"May it please your Honour, immediately upon the Queen's arrival here (Holyrood) she fell acquainted with a new disease that is common in this town, called here the 'New Acquaintance,' which passed through her whole Court, neither sparing lord, lady, nor demoiselle—not so much as either French or English. It is a pain in their heads that have it, and a soreness in their stomachs, with a great cough that remaineth with some longer, with others shorter time, as it findeth apt bodies for the nature of the disease. The Queen kept her bed six days; there was no appearance of danger, nor many that die of the disease except some old folks. My Lord of Murray is not presently in it, the Lord of Liddington hath had it, and I am ashamed to say that I have been free of it, seeing it seeketh acquaintance at all men's hands."

In 1580, again there raged from the middle of August to the end of September a malignant epidemic catarrh; it began with "a pain of the head and feverish heat: some were disposed to sleep, others to watching: presently followed a dry cough, pain of the breast, huskness and roughness of the throat, weakness of the stomach; at last a terrible panting for breath, like dying persons. Though the cough lasted not long, yet the panting for breath continued to the fourteenth day; some sweated, such recovered the thirtieth or fortieth day; they did not expectorate much. With some the disease went off by stool, in others by urine." Here too it is noted that bleeding was a fatal measure. "Though all had it, few died in these countries except such as were let blood of or had unsound viscera." Bleeding is said to have killed two thousand people in Rome.

Then comes an interval of twenty years. But in 1610, "catarrh" prevailed all over Europe. After that influenza is scarcely heard of again till 1658. Perhaps the plague which prevailed during the greater part of the period left no room in men's minds for thought of the lesser scourge.

In 1658, there was an epidemic, an excellent account of which we owe to Thomas Willis, the discoverer of diabetes, who has writ his name for all time on the base of the brain. After an unseasonable summer followed by a healthy winter, the spring came bringing ague and also influenza. "About the end of April, suddenly a distemper arose, as if sent by some blast of the Stars, which laid hold on very many together; that in some towns, in a space of a week, above a thousand people fell sick together. The particular symptom of this disease, and which first invaded the sick, was a troublesome cough, with great spitting, also a catarrh falling down on the palat, throat, and nostrils; also it was accompanied with a feaverish distemper, joyned with heat and thirst, want of appetite, a spontaneous weariness, and a grievous pain in the back and limbs: which feaver, however, was more remiss in some, that they could go abroad, and follow their affairs in the time of their sickness, but complaining in the mean time of want of strength and of languishing, a loathing of food, a cough, and a catarrh." He goes on to say that "such as were indued with an infirm body or men of a more declining age, that were taken with this disease, not a few died of it; but the more strong, and all of an healthful constitution, recovered."

In 1675, there was another visitation which had a still more distinguished historian. Sydenham records that an unusually mild autumn was succeeded, at the end of October, by sudden cold and moisture. Coughs prevailed in greater number than at any other time within the remembrance of the English Hippocrates. "No one escaped them, whatever might be his age or temperament; and they ran through whole families at once." He adds that they were remarkable "on account of the accidental dangers which they brought upon those affected."

In 1720, another highly competent observer, John Huxham, of Plymouth, described an epidemic. "About this time," he says, "a disease invaded these parts which was the most compleatly epidemic of any I remember to have met with; not a house was free from it; the beggar's hut; and the nobleman's palace were alike subject to its attacks; scarce a person escaping either in town or country; old and young, strong and infirm shared the same fate." The distemper, he says, had raged in Cornwall and the western parts of Devonshire from the beginning of February; it did not reach Plymouth till about the 10th. On that day, which was a Saturday, numbers were suddenly seized; the next day "they fell down in multitudes, and by the 18th or 20th of March, scarce any one had escaped it." Huxham's description of the disease is worth quoting:—

"The disorder began at first with a slight shivering; this was presently followed by a transient *erratic* heat, an headache, and a violent and troublesome sneezing; then the back and lungs were seized with flying pains, which sometimes attacked the breast likewise, and though they did not long remain there, yet were very troublesome, being greatly irritated by the violent cough which accompanied the disorder; in the fits of which a great quantity of a thin sharp mucus was thrown out from the nose and mouth. These complaints were like those arising from what is called *catching cold*, but presently a slight fever came on, which afterwards grew more violent; the pulse was now very quick, but not in the least hard and tense like that in a pleurisy: nor was the urine remarkably red, but very thick and inclining to a whitish colour; the tongue, instead of being dry, was thick covered with a whitish mucus or slime; there was a universal complaint of want of rest, and a great giddiness. Several likewise were seized with a most racking pain in the head, often accompanied with a slight delirium. Many were troubled with a *tinnitus aurium*, or singing in the ears; and numbers suffered from violent earaches or pains in the *meatus auditorius*, which, in some, turned to an abscess; exulcerations and swellings of the *fauces* were likewise very common. The sick were in general very much given to sweat, which, when it broke out of its own accord, was very plentiful, and continued without striking in again, did often in the space of two or three days wholly carry off the fever; the urine depositing a copious, whitish or yellowish coloured sediment, but very seldom a reddish one; numbers, however, had great difficulty in making water, whether from their blisters, or from their profuse sweats, I will not take upon me to determine. The disorder often terminated with a discharge of bilious matter by stool, and sometimes by the breaking forth of fiery pimples. You have here a description of this *epidemic* disease, such as it prevailed hereabouts, attacking every one more or less; but still, considering the great multitude that were seized by it, it was fatal to but few, and that chiefly infants and consumptive old people. It generally went off about the fourth day, leaving behind a troublesome cough, which was very often of long duration; and such a dejection of strength as one would hardly have suspected from the shortness of the time; but this chiefly happened where there had been an imprudent and untimely use of the lancet . . . . . On the whole, this disorder was rarely mortal, unless by some very great error arising in the treatment of it; however, this very circumstance proved fatal to some, who, making too slight of it, either on account of its being so common, or not thinking it very dangerous, often found asthmas, *hectics*, or even consumptions themselves, the forfeitures of their inconsiderate rashness."

The last sentence shows that in the eighteenth century, as in the twentieth, the real danger of influenza was the "contempt for the enemy," which has so often brought the sturdy Briton to grief in other spheres.

In 1732–1733, an epidemic occurred of which we have a description by the celebrated John Arbuthnot. He notes that in the accounts he had seen from Germany, France, and some other places, the air in the beginning of winter, especially in November, was more filled with thick and frequent fogs. "Fogs," he adds, "are so usual in this country in November, that there was nothing particular observed about them that I know." He goes on to say:—

"It invaded Saxony and the neighbouring countries in Germany about the 15th of November, and lasted in its vigour till the 29th of the same month. It was earlier in Holland than in England; earlier in Edinburgh than in London. It was in New England before it attacked Britain; in London before it reached some other places westward, as Oxford, Bath, etc.; and, as far as I can collect from accounts, it invaded the northerly parts of Europe before the southerly. It lasted in its vigour in London from about the middle of January, 1732-3, for about three weeks; the bill of mortality, from Tuesday the 23, to Tuesday the 30 of January, contained in all 1588, being higher than any time since the plague. It began in Paris about the beginning of their February, or the 21 of our January, and lasted till the beginning of their April, or the 21st of our March, and I think its duration was longest in the southerly countries. It raged in Naples and the southern parts of Italy in our March. The disease, in travelling from place to place, did not observe the direction, but went often contrary to the course, of the winds."

His description of the symptoms is very interesting, and shows the keenness of his clinical insight. It will be noted that he observed the peculiar fatality of the disease in old people, the extraordinary debility which it left behind, and the manifold "nervous distempers" which followed the attack.

"The uniformity of the symptoms of the disease in every place was most remarkable. A small rigor or chilliness, succeeded with a fever, of a duration (in such as recovered) seldom above three days. This fever was attended with a headache, sometimes pains in the back, thirst in no great degree, a catarrh of thin defluxion, occasioning sneezing, a coryza, or running at the nose; a cough with expectoration of a thin pituite at first, and afterwards of a viscous matter, in which, if there was observed a clear oily matter, it proved generally the case to be mortal; for this clear matter was purulent. These were the most common symptoms; but a great many, during that season, were affected with spitting of blood, pleurisies, and inflammations of the lungs, dangerous and often mortal; in some places, particularly in France, the fever, after six or seven days, ended in miliary eruptions; in Holland, often in imposthumations of the throat; in all, the blood was sizy; and everywhere the disease was particularly fatal to aged people. What was observable was, that the fever left a debility and dejection of appetite and spirits, much more than in proportion to its strength or duration; and the cough outlasted the fever in some more than six weeks or two months. There was, during the whole season, a great run of hysterical, hypochondriacal, and nervous distempers; in short, all the symptoms of relaxation. These symptoms were so high in some, as to produce a sort of fatuity or madness, in which, for some hours together, they would be seized with a wandering of their senses, mistaking their common affairs; at the same time, they had not any great degree of fever to confine them to their beds; but in several who were thus affected, the urine was observed often to change from pale to turbid, alternately, so that there was some fever."

Of epidemics in 1737 and in 1743, we have descriptions by Huxham; the latter he speaks of as "exactly the same with that which in the spring was rife all over Europe, termed the 'Influenza.'" He adds, "that it was much more destructive in the southerly nations than it was here; nay, even in London it increased very greatly the number of burials, rising them, in one week only, to at least a thousand."

There was a severe epidemic in 1762, which was described by Dr., afterwards Sir George, Baker, to whom we owe the elucidation of lead-colic. He notes that a great number of those affected were very slowly restored to health, languishing for months, some even for a whole year, with cough and feverishness—relics of the disease which it was difficult to shake off. Some, after struggling long with impaired health, fell victims to pulmonary consumption. He observed, too, that those persons suffered most severely, who could not obtain a respite from labour; more especially those who worked daily in the open air. "Among this class the pestilence was so violent that it destroyed many of them within four days in spite of remedies."

In 1762, W. Watson, writing to Huxham, speaks of a disease "very epidemic, though not fatal," which had for some time before been very prevalent both in Italy and in Germany. "It is nearly the same disease which was in London in April and May, 1743, and then called *Influenza*, the name applied to it in Italy."

Heberden described a visitation in 1767. It appears to have been of a mild type, for he says of it that "there seems no reason to apprehend any of those lasting ill-consequences from it which attended the sufferers in 1762, many of whom continued in a languishing state for several months and then died; and others complained for two or three years that its ill-effects still hung upon them, and that they had not, in all this time, recovered perfectly from the hurt which it had done their constitution." One might be reading the words of a living physician.

We have descriptions of a very severe epidemic in 1775, by Drs. Fothergill, Heberden, and Sir George Baker, and a number of other physicians. In 1782, the disease raged so fiercely and so widely that the London College of Physicians issued a "public advertisement" to the physicians of Great Britain and Ireland asking for information. The replies were published in the *Medical Transactions* (Vol. III.). Epidemics occurred in 1789–90, and 1803. Then came an interval of nearly thirty years. In 1831, another visitation occurred, which lingered about for several years. In 1837, the Council of the Provincial (now British) Medical Association issued a circular to the members asking for information respecting the origin, progress, and duration of the epidemic, its symptoms and treatment, the atmospheric phenomena attending and preceding it. An elaborate report, based on the information thus obtained, was drawn up by Dr. Robert J. N. Streeten, and published in the *Transactions* of the Association (Vol. VI., London, 1838). Neither the College of Physicians nor the Provincial Medical Association got any nearer a satisfactory solution of the problem of influenza than Molyneux, a seventeenth century writer, who said that the cause of influenza was "something subtle and occult in the atmosphere." What that something is we have yet, as this issue of THE PRACTITIONER shows, to discover.

The most widespread visitation, before the great outbreak of 1889–90 occurred in 1847. In one of Mrs. Carlyle's letters, written at the time, it is unkindly suggested that the term "influenza" had been invented by the doctors to save them the trouble of making a diagnosis. It is to be feared that, even at the present day, influenza, like charity, is a convenient cloak which covers a multitude of diagnostic sins.

NOTES ON INFLUENZA FROM FOREIGN JOURNALS.

VERY little indeed has appeared in foreign journals about influenza for some time past. The *Therapeutic Gazette* and the *Therapeutische Monatshefte* have not contained a single reference to the subject during the last two years; the *Revue de Thérapeutique* had one reference last year, while the *Zentralblatt für innere Medezin*, a great collector, had less than ten references in 1904 and 1905 together, all of which were on points of small interest and importance. The encyclopædic article by Prof. O. Leichtenstern[1] of Cologne University, which appeared in Nothnagel's *Practice*, still holds the field as an authoritative statement of the position of affairs, although written in 1898. In the English edition, published in 1905, Dr. Grünbaum of Liverpool states, in his editorial note, that "the seven years, which have passed since the publication of Leichtenstern's book, have not seen many essential additions to our knowledge of influenza."

The first real pandemic occurred in 1580, spreading from east to west and south to north. There was a decided pandemic in 1781-82, and a most pronounced pandemic period from 1788-90. Two or three pandemic outbreaks, which were remarkably intense and extensive, occurred between 1830 and 1833, and another in 1847-48. The most extensive and important of all the pandemic outbreaks was in 1889-90 when the disease extended all over the world.

Leichtenstern was of opinion that the small outbreaks of catarrhal fever and cough epidemics, which are usually classed as la grippe, or influenza, are not identical with the true influenza, which we learned to appreciate for the first time in the winter of 1889-90, but he looked to the bacillus of R. Pfeiffer as a certain means of identification. If, however, all the many outbreaks and epidemics, tabulated by Hirsch, prove to be one and the same species of influenza, the affection is exceedingly variable and protean in its epidemiological features.

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[1] "The irony of fate decided that he should die of croupous pneumonia, following influenza, on February 23rd, 1901, at the too early age of fifty-four years."—[EDITOR'S NOTE.]

Treatment of Influenza. General prophylaxis is regarded as being practically impossible, for the disease is so intimately connected with human intercourse and commerce, and its germs are carried so quickly all over the world by numbers of people. No effective quarantine can be put in force against an influenza epidemic, except in the case of shipping, when landing can be prohibited from any vessels having influenza patients on board. It is stated that Australia escaped an epidemic in this way in 1891. It is useless to prohibit large meetings, exhibitions, fairs, markets and pilgrimages. Schools are undoubtedly foci of infection, but it is of no avail to close them, as the children are equally exposed to infection, remaining at home or playing in the streets.

Individual prophylaxis is equally futile, for though, in local epidemics, anyone may escape at the cost of infinite trouble in securing complete isolation, or by instant flight to some unaffected district, "pandemic influenza will pursue and surely overtake the fugitive." In sporadic cases, or when a mild epidemic is abroad, a patient can be strictly isolated from the rest of the household, and thorough methods of disinfection rigidly carried out, but these means fail altogether in the presence of a pandemic. They have, however, proved effective and of use in the case of prisons, lunatic asylums, hospitals and barracks.

It was advised, in order to prevent infection, that disinfecting lotions should be used for the mouth and throat several times a day, and the nose treated with irrigations, sprays, or menthol snuff. Oil of Eucalyptus has been largely used in England for inhalations ; creolin, camphor, oil of peppermint have been used by others. No harm can be done by cleansing the mouth and throat, but applications to the nasal mucous membrane may set up irritation and hyperæmia, and so actually increase the chance of infection by providing an excellent nidus for the germs in the nasal mucus.

Other panaceas, such as cod-liver oil (Ollivier) and sulphide of calcium (Green), sought to confer immunity on the body, but these have been given up.

It was asserted by Gruser of Bonn that "quinine given at the proper time, and in proper doses, is capable of preventing the outbreak of influenza." A troop of Bonn Hussars had a daily dose of 7½ grains of quinine in whisky for three weeks, and suffered less in comparison with other troops. Wider experience of the method, however, points to the conclusion that the "prophylactic action of quinine is exceedingly questionable."

One observer, Mosse, stated that he had proved the immunising action of quinine by experiment, but as Pfeiffer himself has not been able to produce influenza in animals, this so-called proof does not go for very much.

Quinine was very largely used in France and Russia for the treatment of the disease, as being a bactericidal agent. It was so greatly praised as to become the popular panacea, but Leichtenstern found that, although it showed its antipyretic properties in the fever of influenza, and influenced some neuralgias during the convalescent stage, it did not exert the least effect upon the influenza process as such. Cases, treated with large doses of quinine, always felt worse (increased headache, etc.) than those not treated by quinine.

Reviewing the large amount of information contained in the official reports from the whole of Germany, Friedrich says that brilliant results were obtained from quinine by some, while others were very much disappointed.

Treatment by purgation, largely employed in the eighteenth century, was revived. Schuster and Dumas gave calomel in purgative doses, Sajorski and Morles large doses of castor oil, and both claimed to cut short the attacks. It is suggested that only the purely gastro-intestinal form of the affection would benefit by laxatives and intestinal disinfectants, and the occurrence of severe hæmorrhagic forms of influenzal enteritis makes it necessary to be cautious in giving purgatives. This of course does not apply to the relief of constipation at the onset of the disease.

Many preparations have been vaunted as "specifics," purely on empirical grounds. Among these are chloride of ammonium (much praised in France), benzol, benzo-naphthol, carbolic acid, creasote, creolin, turpentine, ichthyol, balsam of Peru, cinnamic acid, tannin, iodide of potassium, perchloride of mercury, double chloride of gold and sodium, sulphide of calcium, sulphocarbolate of sodium, carbonate of potash, aconite, gelsemium.

As the disease ends favourably and spontaneously in a few days, as a rule, the "credulous therapeutic optimist" is quite confirmed in his view of *post hoc, ergo propter hoc.* Happiest of all are the homœopaths, with a specific remedy that is infallible, but they must share their triumphs with "nature doctors," "magnetopaths," followers of Kneipp, vegetarians, "Jæger" enthusiasts, and Christian scientists.

Unfortunately, there is at present neither a prophylactic nor a specific for influenza, and the only hope lies in the production of an "influenza-serum," of which there does not seem to be much probability. Goldschmidt and Althaus have suggested, the former on the strength of some observations in Madeira, that vaccination confers a protection against influenza.

As there is no specific remedy, the treatment of influenza resolves itself into symptomatic treatment. This covers a large area owing to the protean character of the symptoms, and so affords plenty of scope for skill in the management of patients as well as in the use of drugs; consequently, every case must be dealt with on its merits. Certain broad lines of treatment are set down. The greater number of cases require no more than rest in bed, limited diet and attention to hygienic measures affecting the comfort of the patient. Warmth is practically unanimously admitted to be a necessity. Cold applications are badly borne and increase the discomfort. Diaphoresis may be encouraged at the onset of the attack by taking a hot bath, a Turkish, or Russian bath, by wrapping the patient in blankets, and giving hot demulcent drinks. Leichtenstern doubts the value of a vapour bath at any stage of the disease, for the fever is only increased, the headache and sweating made worse, and the patient's general condition brought down. Sponging with lukewarm water, with or without spirit, lukewarm baths, the wet pack and sitz-baths are useful at times. The cold bath is contra-indicated owing to the cough, diffuse bronchitis, dyspnœa, general sweating and the weakness of the heart so often present, in addition the headache, neuralgia pains, and nervous prostration are not improved, but often increased. Ice-bags to the head are not liked by patients; they give no relief and increase the pain.

The drugs, which are of value, are certain sedatives and anti-neuralgics; these produce favourable effects, although possessing no specific action on the influenza itself. Of these, Leichtenstern commends antipyrin, as first among them, and goes on to say: "Only lack of experience, or preconceived prejudice, can doubt that antipyrin prescribed at the right time, in proper doses, is often of great use for the severe neuralgias and myalgias and the general hyperæsthesia and insomnia, both at the acme of the disease, as well as at its onset, and often gives, at least transitory rest, to the much-burdened patient without doing him the least harm." Phenacetin ranks next to antipyrin, but antifebrin is of much less value. The antineuralgic and sedative action of salicylic acid has been found of the utmost value by many observers. Schäffer praises benzoate of soda for its immediate effect in relieving pains.

There have been many combinations of antipyrin and salicylic acid produced under various names, salipyrin, migrainin, salophen, etc. As each component is of use the combination produces the same result.

These drugs should be given in moderate doses every two hours. High fever can be brought down by doses of 5 to 7½ grains of phenacetin, or 5 to 15 grains of antipyrin, and the temperature must be taken every two hours during the time the administration is kept up, so that no more may be given than is necessary. A temperature, which is not reduced by 7½ grains of phenacetin, 15 grains of antipyrin, or 30 grains of quinine, is not suitable for treatment by drugs.

Later additions to this list are pyramidon and acetylsalicylic acid (patented in Germany under the name of aspirin). Of these two, and in fact of the whole preceding list, the last mentioned will be found the safest and most efficacious. It is usually given in cachets in doses of 10 grains, but an easier, and at the same time more effective, method of administration is the following:—

℞. Acid. Acetyl-salicylic - - - gr. x.
Sodii Bicarb. - - - - gr. x.
Aq. Chloroformi - - - ℥j.

Misce. 2^{dis} horis sumend.

Note for Dispensing.—Mix the two powders with the water in a mortar, stir up until all effervescence has ceased, and then decant into the phial.

In the experience of those who have used this method of administration no irritation of the stomach has been caused by the drug, and the therapeutic effect appears to be obtained more quickly and with greater certainty.

For obstinate neuralgias, occurring during convalescence, quinine is of value. Leichtenstern endorses the opinion of Curschmann and Bäumler to the effect that "the tried drug quinine is in many cases of subsequent neuralgia more effective than its modern competitors, and is frequently useful when they have failed."

For the relief of the sharp pain of pleurisy, spasmodic cough, and dyspnœa, which are beyond the action of the sedatives already mentioned, opium must be used. There need be no fear of its "paralysing action on the heart," or of engendering the morphia-habit. Under the influence of an hypodermic injection of morphia the pleuritic pains abate, the cough gets less, the respiration becomes slower and deeper, and, as a result, the pulse improves. Leichtenstern instances the case of a physician, who suffered from cardiac influenza, and had long and severe anginal attacks. Injections of morphia relieved these, quieted the breathing, and restored the pulse, which was previously imperceptible.

The ordinary insomnia of influenza should not be treated by morphia, or hypnotics (sulphonal, trional, etc.), as it passes off with the disease, but these may become necessary when the insomnia gets severe.

Alcohol is found usually to increase the headache, but it should be allowed to those accustomed to its use, and it must be given in the case of cardiac weakness. It is not a prophylactic, as was alleged by French physicians, who recommended it to be taken as hot drinks.

Finally, every care must be taken during convalescence to avoid catching cold, and escape the risk of pneumonia and relapses. The patient must be kept in bed as long as possible.

More Germanico, the interest in the treatment of the case seems to cease when the acute stage is past, though influenza is certainly one of the diseases, in which the period of convalescence needs careful treatment and dieting. This loss of interest may perhaps be more apparent than real, owing to the fact that the German patient never stops long upon low diet, if indeed he ever takes it, for many physicians never order it even in typhoid and rheumatic fever.

Notes by the Way.

Influenza.

ABUNDANT as have been our opportunities of studying influenza during the last sixteen years, we have not yet learnt how to prevent its invasion, or how to stamp it out when it has got a footing in our midst. The devotion of a whole issue of THE PRACTITIONER to the disease therefore needs no excuse. In accordance with the usual plan of our special numbers—of which we may be allowed to say the medical profession both in this country and in America has shown a warm appreciation—an endeavour has been made to consider the subject from every side, and the names of the writers who deal with the various aspects of this "multifaced" (if we may borrow the epithet which Southey applies to Satan) scourge are sufficient guarantees of the scientific value of their contributions. Professor Clifford Allbutt reviews the whole subject, and indicates the salient points of the other writers. Little more, therefore, need be done here but to show the comprehensive manner in which the disease is dealt with by a brief indication of the contents. Sir Richard Douglas Powell deals with important clinical features. Sir William Broadbent's note on the value of quinine as a prophylactic embodies the result of a particularly ripe experience ; Sir Dyce Duckworth calls attention to a matter of great practical importance, the risk of infection in railway carriages. To this risk invalids on their way to the South of Europe are especially exposed, as in them the bacillus finds an easy prey. Sir John Moore, Dr. Samuel West, and Dr. Hector Mackenzie treat exhaustively of the pulmonary manifestations of influenza ; Dr. Harris deals with the nerve affections, and Dr. Claye Shaw with the mental disorders which follow in its train. Dr. Norman Dalton discusses the intestinal forms of the disease, while Mr. Armour points out the special features of appendicitis following influenza. Dr. Cowan, Dr. StClair Thomson, Mr. Cheatle, and Dr. Lyle deal respectively with the heart, throat, ear, and eye complications. What is known of the bacteriology of influenza is clearly set forth by Dr. d'Este Emery. His conclusion shows how very dim and uncertain is our knowledge of the causation of the disease. Dr. Newsholme, who considers influenza from a public health standpoint, expresses the comforting view that the scourge is diminishing in virulence, and that it will in a few years again become an insignificant quantity in our vital statistics.

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The Open Window and Influenza.

DR. MACKENZIE holds that the best prophylactic against influenza is fresh air, which he also regards as a sovereign remedy in the treatment of both acute and chronic manifestations of the disease in the respiratory tract. He says the stuffy and ill-ventilated hotels, in which people generally have to live who travel in search of health, are, as it were, forcing beds for the disease, and he denounces the people who insist on closed windows in trains and hotels as among the causes which make it spread as it does. He throws out a suggestion that a hotel or sanatorium for non-tuberculous patients convalescing from influenza should be established in one of our own south coast health resorts, and run on open-air lines. We agree with him that such an institution would be likely to do much to promote the rapid and complete recovery of its visitors. We may, perhaps, be allowed to hint a word of warning against the tendency to make a fetish of the open window which now threatens to carry the profession beyond the teachings of common experience. We remember beds in hospital wards, which, by being under a window, bore the significant name of "pneumonia beds." We recall a strenuous resident medical officer who, going through the wards at night, found them "stuffy," and ordered all the windows to be opened wide, the result being a plentiful crop of "respiratory affections" of one kind or another among the patients. The fact is that in the matter of tolerance of cold air, there are, as in regard to drugs and foods, differences even in healthy people which must be taken into account. Let us have fresh air by all means, but let the wind be tempered to the shorn lamb.

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The State of the Medical Profession.

DR. J. D. RAWLINGS, of Dorking, has sent us the following very interesting letter in reference to some remarks on the subject which appeared in THE PRACTITIONER for November :—

"It has occurred to me that some of your readers may conceivably be interested to know the views of a general practitioner, who is entirely satisfied with the profession both as a means of livelihood and as an occupation, and who attributes his giving satisfaction to the fact that he thinks he has discovered how to deal with some of the difficulties which beset the profession. I am one of seven active practitioners in a town whose population is 7,670. There is no good class building going on, and each of the surrounding villages has its own doctor.

"Personally, I hold very strongly that all the main causes of our troubles lie within the profession itself, and may be summed up briefly as *want of grit.* We have not the grit collectively or individually to hold our own, and take the position in the world which we might have if we deserved it. If collectively we had the grit to combine, there would be an end to all our grievances. We should have the rest of the world in the hollow of our hand, and could make our own terms with poor law officials, clubs, anti-vaccinators, dispensing chemists, and even quacks. Combination, however, is, as you say, impossible, and it is useless to dream of what it might lead to. But let us look the fact squarely in the face that we, the Medical Profession, have proved ourselves unable to do what the British working-man has done with complete success all over the kingdom. There is, nevertheless, a vast amount that might be done by medical men individually, were it not for this unfortunate defect in the medical character. Indeed, I believe that if we were all men instead of the weak-kneed, invertebrate creatures that most of us are, there would be little need for combination. The object of my letter is to plead for individual action which is possible, rather than for collective action which is impossible.

"Let us take our principal grievances seriatim, and see what can be done for each of them by individual action.

"1. We are bullied and underpaid by guardians and clubs.

As things are at present, very many of us are obliged to take contract work on the terms offered us ; but it is quite unnecessary to tolerate bullying by the secretaries, or insolence from the patients, or the latter coming to the surgery at irregular hours, or sending for the doctor when they might come to the surgery, or sending in the night when they might do so in the daytime, or on Sunday when they might do so on a week-day.

"During the first two years that I was in private practice, my work consisted largely of club and parish work. During the latter part of this time, I firmly refused to treat any patient who did not comply with my regulations as to hours, etc. My methods very quickly became known, and by the time I was able to give up contract work, the annoyances and waste of time connected with it were reduced to a minimum, and the amount of work was not so very disproportionate to the emoluments. But how many men in the profession have the grit to adopt this method effectively ? When I suggest it to anybody who is grumbling about contract work, I am generally met with the reply : 'Well, after all, it is less trouble to give a bottle of medicine than to make the patient come at another time.'

"2. We are bilked by a certain proportion of our patients. Each man can almost entirely stop this for himself by sending out his bills quarterly, or at the end of each attendance. In my experience, a patient who intends to bilk does not generally come again after he has received a bill. If he does, and one takes him for one of the gentry who can pay but won't pay, one can remind him that his bill is unpaid. By this means I have reduced my bad debts to a negligible quantity. But how many of us have the grit to do this ? Instead, we allow bilkers to run on in our debt for a year or so before we even send a first bill. When a new patient comes to me, I always ascertain whether he has been attended by any other doctor in the town, and if he has, I communicate with the doctor. I do so partly out of courtesy to the other doctor, and partly in order to learn whether the patient has paid his fees. When a patient has treated me badly, I send a caution about him to all my confrères, and I think it says a good deal for the success of my methods that I have not had occasion to send one of these cautions for two or three years. I am not popular with the bilkers. But again I ask, how many men in the profession take these simple steps for their own and their friends' protection? I may mention that I have never yet sued for a debt.

"3. Our charity is imposed upon by the well-to-do. When an apparently unsuitable person presents himself for treatment at the local hospital, I find the simple little question, 'Are you in need of charity?' has a wonderful effect. My own outpatient department is entirely purged of this class of person. The news that such a question is liable to be asked and pressed home, spreads like wildfire, and the fraudulent cannot face it. But how often does a medical man take this matter into his own hands? Instead, we grumble at the apathy of the lay committee in the matter, and content ourselves with emulating it.

"4. Our prescriptions are carefully preserved and used for subsequent ailments for which they were not intended, they are lent to friends of the patient to whom they were given, and undoubtedly, I think, sometimes reach the hands of patent medicine vendors. Why give prescriptions? It is quite unnecessary. On the few occasions when it is necessary to give a prescription, why not write across it 'Not to be dispensed after——.' There again, each man can stop the grievance for himself, as I have done. I do not think I give a dozen prescriptions in a year. I order all medicine from the chemist, who sends it to the patients and charges me for it.

"5. We are insulted and flouted by 'anti-vaccinators.' I have but little hesitation in saying that if any considerable proportion of the profession had adopted an active and militant policy in this matter, and done their obvious duty (an unpleasant and troublesome duty, I admit) to themselves and the public, this handful of cranks and uneducated bigots would never have attained to the importance that they have done. I have proved by my own efforts in this direction, that one medical man, acting alone, may do an appreciable amount of good by taking up the gauntlet so often thrown at our feet by these persons. Instead, we either pretend not to see it or divide it, professing to consider our slanderers unworthy of serious attention.

"6. We are exploited by commercial undertakings for the enrichment of the laity at our expense. The most glaring example of this, to my mind, has occurred quite recently, when, in order to provide copy to the morning papers during the silly season, a number of us wrote letters to these papers advertising to the world at large what downtrodden worms we medical men are. Could anything excel this proceeding in puerility and fatuousness?

"I much regret, Sir, that I have been unable to express my views without giving to my letter an apparent vein of egotism. It is due, not to conceit, but to my desire to emphasise the fact that my opinions are based on my own personal experience, and not on theory."

We heartily endorse Dr. Rawlings's statement that a large proportion of the troubles which doctors complain of are of their own making. If, as he says, they showed more "grit" in their professional dealings, their charity and good nature would not so often be abused. But the pity of it is, that in places, where competition is severe, only a man of exceptional gifts can, under existing conditions, afford to take the strong line that will enable him to earn the respect of the public without detriment to his pocket.

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False Brethren.

If the doctor is often an enemy to himself, he still more frequently finds worse enemies in the household of the profession. Were it not for false brethren, the doctors would have little to fear from the public. The point is illustrated by the following letter from a correspondent who signs himself M.D.:—

"*Re* doctor's incomes. A recent experience of mine may be of some interest in illustrating (1) the attitude of the public towards the doctors, and (2) the attitude of the doctors to one another.

"A local tradesman brought his son to see me. The boy was suffering from whitlow, which I opened under ethyl chloride locally. He subsequently paid me nine visits. As the father was a small shopkeeper, I sent in what I considered was a moderate account, for 22s. 6d. I was greatly astonished on hearing from this man, several months later, that he had no intention of settling my account, as it was at a much higher rate than he had ever paid for medical attendance, and that he could get a doctor from — , and, as a fact, a doctor had been over from that neighbourhood—about three miles away—for a fee at a lower rate than I had been charging.

"Sir, your advice to medical practitioners to combine is excellent; but is such combination possible as long as 'blacklegs' do not break the law? The futility of such an attempt at combination was shown quite recently in this district. The local lodge of Foresters required a surgeon; the local medical men in the district combined to refuse a rate of payment under 5s. per head per annum; the result was that a medical man outside the district accepted the appointment, which he would not have got otherwise, at 4s. per head.

"As a profession, if we are to find a remedy for our ills, the only way, it appears to me, is for us to adopt trade union principles; and the General Medical Council should be powerful enough to fix a 'minimum living wage,' and to punish 'blacklegs' who might be willing to give their services below it. We might then, as a body, be able to resist being exploited by lay committees and sweated generally by the public."

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The General Medical Council.

It will be noted that our correspondent looks to the General Medical Council as the body which should redress the wrongs of the profession. He evidently knows what so many of those who air their grievances in the Press, medical and lay, do not know, that the Council at present has no power either to fix a scale of fees or to punish practitioners for "underselling." To judge from their election addresses most of the candidates, who recently offered themselves as direct representatives, believed that the Council has the power to do these things. Its duties and such powers, as are conferred upon it by the Legislature, relate solely and exclusively to the putting an official stamp on legally-qualified practitioners which shall enable the public to distinguish them from the unqualified, to maintaining an adequate standard of medical education, and to purging the *Medical Register* of the names of persons adjudged guilty of "infamous conduct in a professional respect."

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"Infamous Conduct."

Now what is "infamous conduct in a professional respect"? The Court of Appeal has given us the definition. In 1892, that tribunal laid it down that "if it is shown that a medical man, in the pursuit of his profession, has done something with regard to it which would be reasonably regarded as disgraceful or dishonourable by his professional brethren of good repute and competency, then it is open to the Council to say that he has been guilty of infamous conduct in a professional respect." Legal definitions are always so elastic that it might seem they were intended to provide matter for disputes as to their interpretation. It is certain that most men of "good repute and competency" regard it as disgraceful and dishonourable to "cut their prices" like rival grocers. Yet we have it on the authority of the President of the Council, who must be held to know exactly what the law means and intends, that the Council cannot "say a word on the matter of rates of pay, or hours of work, or disputes with employees." Nor, according to Dr. MacAlister, can it do anything to put down quackery; the British public is as free to seek counsel of false oracles in medicine as in theology, or in anything else, always saving and excepting law, which the lawyers have taken care to secure as a *hortus inclusus* to be kept sacred from profane feet.

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The Reform of the Council.

For this difficulty it will be said by the irresponsible agitator, there is an easy remedy: In the words of Hamlet, "Reform it altogether." Taking "it" here to mean the General Medical Council, we would ask who is to reform it? There appears to be a widespread belief that the profession itself could do all that is needed, if it had the election of the Council in its hands. Of what a council wholly composed of direct representatives would be, a fairly correct idea may be formed from the experience already gained of direct representation. Can it be seriously maintained that the men who have so far been chosen by the profession have, as a whole, fulfilled the anticipation of the Royal Commission of 1882, which said "no reason to suppose that the members elected by direct representation will be less eminent than those nominated either by the Crown or other bodies"? There have been one or two notable exceptions, but these have been consultants, a stigma from which candidates in the recent election took great pains to clear themselves. Frankly, what has been done by direct representatives does not inspire us with confidence that they will be able to achieve much in the future.

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Amendment of the Medical Acts.

EVEN if the Council contained a sufficient number of direct representatives to outvote—if they happened on any occasion to be unanimous—the nominees of the Crown and the representatives of the universities and corporations, the Council could not go beyond the powers conferred upon it by the Legislature which, as has been said, are strictly limited. To enable it to fix scales of remuneration, punish blacklegs, and put down quackery, these powers would have to be greatly enlarged. For this purpose the Medical Acts would have to be amended to an extent that would amount to an entirely new measure, which among other things would abolish free trade in the treatment of disease and give doctors a highly profitable monopoly. The suppression of quackery would unquestionably be for the benefit of the public, but the public is on this matter incurably blind to its own interest. And it is the people which elects Members of Parliament, and the Legislature can after all only express the will of the people. If the late Parliament could not be got to undertake the amendment of the Medical Acts, what chance is there of such measure finding any effective support in the present one, in which anti-medical influences are rampant and the profession scarcely represented at all? It is difficult enough to get Parliament to make the laws for the good of the nation; it is hopeless to expect that it will do so for the benefit of a class, especially one which, in a political sense, may safely be left out of account.

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The Remedy is Combination.

THE remedy lies with ourselves, and it is expressed in a single word *combination*. The medical profession holds a position of impregnable strength, for though the people run after quacks, they cannot dispense with the qualified doctor, even if he be regarded as an evil, he is felt to be a necessary evil. There are certain ritual observances in which only a doctor recognised by the law can officiate as the minister, and no one can, without inconvenience to survivors, die in the ordinary course of nature without the last consolations of orthodox medicine. If, therefore, the profession would combine for the defence of its interests, it could enforce equitable treatment from the public. It is, of course, difficult for a large body, the interests of whose members are different and often conflicting, to combine for any purpose, but there are distinct, though faint, signs that the advantages of union are beginning to be appreciated. For our own part we look to the growth of a feeling of solidarity based on fundamental community of interest, and to a higher standard of professional honour rather than to trade unionism to make medical combination effective. The idea of trade unionism is repellent to men who do not look upon their profession as a trade, and the application of its methods to practitioners who were only using the rights which the law gives them has not been conspicuously successful. The disputes with friendly societies have not been edifying, and in one or two cases the self-appointed representatives of the profession have been beaten not only in the fight, but, what to our mind is much worse, in the argument. We must seek to persuade rather than to coerce brethren whose need or whose greed leads them astray into devious courses. We must not boycott except for such offences as really tend to bring discredit on the profession; with these the General Medical Council may be left to deal. It may be added that even a perfectly working system of combination cannot bring about a medical millennium. The battle will still be to the strong, and the race to the swift. No organisation that ever existed or will exist can do more for a man than remove a few stumbling blocks from his path. But his fortune can only be made or marred by himself.

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The Punishment of Hospital Abuse.

ONE of the difficulties of dealing with the abuse of hospital charity by the well-to-do is that there is no punishment for the offence. It was proposed in THE PRACTITIONER several years ago that such cases should be made punishable as actual or attempted fraud; but the suggestion has not borne fruit. The more's the pity, for we are still of opinion that the plan would be at once practicable and effective. It is not always possible to bring the offender to book so sharply as was once done by Velpean. One day the Marquis de —— presented himself in the famous surgeon's consulting room. Velpean after examination told the patient that an operation was urgently needed. On learning that the fee would be £200, the Marquis said: "Very well, I will put my affairs in order and you shall hear from me in a few days." A week or so later Velpean found among the new patients at the hospital one presenting exactly the same symptoms as the Marquis. Struck by the similarity he looked at the patient more closely, and recognised his man, through an elaborate and skilful disguise. Without betraying any suspicion Velpean asked his name and occupation; the replies showed that the Marquis had borrowed the name and identity of his valet. The operation was *duly* performed and was successful. When the patient was out of danger Velpean asked him if he was satisfied. The reply was an effusive expression of gratitude which the patient said he wished he had means of proving. Bending over him Velpean whispered in his ear: "M. le Marquis, it is easy for you to do so. Send to-morrow a donation of £400 to the hospital funds." On the discomfited patient attempting a remonstrance Velpean said firmly: "I will allow neither excuse nor delay. If the money is not forthcoming within forty-eight hours I will send an account of the whole affair, with your name, to the newspapers. The story will amuse the public." The money was paid, and it may be hoped that the Marquis was cured of his meanness as well as of his malady.

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Competitions.

THE results of the November Competitions are as follows:—The best essay was sent by Dr. F. Fowler, of Bournemouth; and the three best cases by Dr. D. T. Barry, of Cork. Cheques have accordingly been sent to these gentlemen.

Practical Notes.

THE TREATMENT OF INFLUENZA.—As soon as it is recognised that a patient is suffering from influenza, isolation should be insisted upon at once, and young children and old persons should be guarded against all possible source of infection ; the latter being especially liable to serious complications and sequelæ. The patient should be at once ordered to a warm bed, be rigidly confined there, and kept quiet until the temperature becomes normal, and from the very first the sick room should be thoroughly well ventilated. Dr. J. F. Goodhart says, "To go to bed, to take plenty of light liquid nourishment, and some liquor ammonii acetatis every few hours, are sufficient, in most cases, to induce a quick recovery." It is safest to thoroughly disinfect any secretion which may come from the nose and the bronchial tubes, and this may best be done by the use of carbolic acid, one part in twenty. The mouth and nose should be systematically and frequently disinfected by antiseptic inhalations, such as oil of eucalyptus or thymol, sprays and washes.

Sir J. W. Moore recommends the local use of quinine and resorcin. A good antiseptic gargle may be made by suspending one grain of sulphate of quinine in a wineglassful of cold water. The following is a useful mouth and nasal spray :—

R. Resorcin - - - - - gr. lxxx.
Glycerini - - - - - - ℥ss.
Aq. Rosæ - - - - ℥viiss.
Misce.

At the outset, the bowels should be opened by a saline purge, such as the effervescing citrate of magnesia or sulphate of soda, or a small dose of calomel, and at night five to ten grains of Dover's powder may be prescribed, and a draught consisting of the following :—

R. Salicini - - - - - gr. xij.
Liquoris Ammonii Acetatis - - - ʒiv.
Aq. Camphoræ - - - ad ℥i.
Ft. Haust.

The patient should be given a supporting diet at first, milk and "egg albumin water"; the latter may be prepared by whipping up the whites of three or four eggs, to which may be added a pint of cold water and some cinnamon as a flavouring agent. Egg-flip may be given later. If a stimulant is required, white wine whey may be used with equal parts of unflavoured egg albumin water. Beef-tea and chicken-broth may be also given separately, or mixed in equal quantities. Later, eggs and chicken, sweetbreads, fish, and finally roast joints, but, as Sir J. W. Moore reminds us, in attempting to draw up a bill of fare for a fastidious influenza patient, we should always remember that "what is one man's food is another man's poison." If the pulse is weak, stimulants are indicated; of these a good port wine and champagne are undoubtedly the best. During the course of the disease, special symptoms, which may be referred to either the *respiratory* or *gastro-intestinal tracts*, or *the central* or *peripheral nervous system*, may arise, and which, in either case, usually call for special medicinal treatment. Under these varying conditions, the following prescriptions may be found to benefit the patients :—

In the early stage of the disease, the following will be found useful :—

R. Quininæ Salicylatis -	gr. xvi.
Acidi Nitrici Diluti - - -	♏xvi.
Tincturæ Aurantii - - -	℥i.
Syrupi Tolui - - - -	ʒss.
Aq. Dest. - - - ad	℥viii.

Misce. Ft. Mist.

Two tablespoonfuls to be taken three times daily.

Dr. Burney Yeo says that the remedy, which is most deserving of confidence in the treatment of influenza, after two or three days of salicin treatment, is *quinine*. It seems to be really an anti-toxin in this disease. Professor J. Tessier, of Lyons, compares its action in influenza with that of sodium salicylate in acute rheumatism, and regards it as the most valuable medicine we possess for the treatment of it. If the treatment by quinine is persevered with, there will be much less cardiac weakness, and fewer of the serious sequelæ which so frequently

The best and most efficacious way of giving quinine will be found to be as follows:—

Dissolve from one to three grains of sulphate of quinine in ten to twenty grains of citric acid, and add the solution to a mixture of ammonium carbonate and potassium bicarbonate in sufficient quantity to rather more than neutralise the citric acid. This dose should be repeated every three or four hours. If profuse and exhausting sweatings occur in the afternoons or evenings, five grains more of quinine sulphate, dissolved in lemon-juice, should be administered about five p.m.

If, however, a patient is unable to take quinine, Packard recommends the following as an efficient substitute:—

R. Strychninæ Sulphatis - - -	gr. $\frac{1}{45}$.
Cinchonidinæ Salicylatis - - -	gr. ij.

Misce. Ft. pil.

One pill to be taken every four hours.

In order to relieve the bronchial catarrh, which so often troubles the patient, the following medicine will prove of service:—

℞. Ammonii Chloridi - - -	ʒij.
Tincturæ Sumbol. - - - -	ʒvj.
Extract Glycyrrhizæ Liquidi - -	ʒiv.
Spiritus Ammoniæ Aromatici - -	ʒiv.
Aq. Chlorofor. - - - ad	℥xij.

Misce. Ft. Mist.

Two tablespoonfuls to be taken three times daily.

Forchheimer states that the ordinary bronchitis is satisfactorily controlled by the following:—

℞. Sodii Benzoati - - - -	ʒij.
Aq. Menth. Pip. - - - -	℥iv.

Misce. Ft. Mist.

One tablespoonful to be given every two hours to an adult.

If the cough is hard and troublesome, with a scanty tenacious expectoration, the following may be prescribed:—

℞. Apomorphinæ Hydrochloridi - -	gr. i.
Morphinæ Hydrochloridi - - -	gr. $\frac{3}{4}$.
Acidi Hydrochlorici Diluti - -	ʒss.
Syrupi Aurantii - - - - -	ʒiv.
Aq. - - - - - ad	℥iv.

Misce. Ft. Mist.

One or two teaspoonfuls to be taken at a time for a dose.

Simson recommends the following mixture for the relief of the cough :—

R.	Liquoris Morphinæ Hydrochloridi	ʒi.
	Acidi Hydrobromici Diluti	ʒi.
	Chloroformi	♏iij.
	Tincturæ Limonis	ʒi.
	Syrup.	ad ℥iss.

Misce Ft. Syrup.

A teaspoonful to be taken when necessary.

Sir William Whitla suggests the following medicine for the relief of influenzal pneumonia :—

R.	Ammonii Carbonatis	ʒiv.
	Tincturæ Cinchonæ	℥iss.
	Spiritus Ammoniæ Aromatici	ʒiv.
	Decoct. Cinchon.	ad ℥xij.

Misce. Ft. Mist.

Two tablespoonfuls with a tablespoonful of lemon-juice in effervescence every four hours. At the same time, cardiac stimulants, such as brandy, strychnine, combined with digitalis and oxygen inhalations, are required.

It is well known that cardiac asthenia often appears during an attack of influenza, and often continues after the attack has passed off; it calls for special treatment. Prolonged rest in bed in a well-ventilated room, or, if the weather is warm, on a couch, in the open air is essential, and the following prescribed :—

R.	Quininæ Sulphatis	gr. i.
	Acidi Sulphurici Diluti	♏iij.
	Tincturæ Ferri Perchloridi	♏v.
	Tincturæ Nucis Vomicæ	♏v.
	Magnesii Sulphatis	gr. xx.
	Inf. Quass.	ad ℥i.

Misce. Ft. Mist.

Two tablespoonfuls to be taken three times daily.

For the severe gastric pain and vomiting, which sometimes occur during an attack of influenza, Huchard recommends the following mixture :—

R. Sodii Bicarbonatis,	
Magnesiæ,	
Bismuthi Salicylatis - - -	āā gr. v.
Misce. Ft. Pulv.	

Three to five powders to be taken daily.

Hydrocyanic acid should not be prescribed for the purpose of relieving vomiting, as it is somewhat depressing to the heart.

For the intense headache and pains in the limbs and back, Sir William Whitla prescribes Antipyrine, 5 grains, with Citrate of Caffeine, 2½ grains, to be taken at once, to be followed by half the dose, every hour for six or eight doses : this, he says, invariably gives speedy relief. One of the most troublesome symptoms of influenza is persistent and severe headache. This may be relieved by the following :—

R. Phenazoni - - - -	gr. x.
Caffeinæ - - - -	gr. ij.
Spiritus Ammoniæ Aromatici - -	ʒss.
Tincturæ Cardomomi Co. - -	ʒss.
Aq. Dest. - - - ad	℥iss.

Three tablespoonfuls to be taken when required.

If neuralgia is severe, the following draught may be prescribed :—

R. Caffeinæ Citratis - - -	gr. i.
Phenacetin. - - - -	gr. vi.
Muciliganis Acaciæ - - -	ʒi.
Aq. Menthæ Pipertiæ - - ad	℥i.
Misce. Ft. Haust.	

Or the following :—

R. Phenazoni - - - -	gr. xv.
Tincturæ Gelsemii - - -	♏xx.
Aq. Chloroform. - - ad	℥ij.
Misce. Ft. Haustus.	

One-fourth part to be taken every second or third hour until the pain ceases.

For the troublesome nasal catarrh, which frequently accompanies influenza, one of the following remedies may be used :—

R. Acidi Borici	gr. xxv.
Menthol	gr. xxx.
Bismuthi Oxychloridi	gr. xxv.
Lycopodii	ʒiv.

Misce. The snuff to be used frequently.

Or,

R. Menthol	ʒi.
Paraffini Liquidi	ad ℥i.

Misce.

This may be locally applied into each nostril with a good camel-hair brush.

For use during convalescence, Sir J. W. Moore recommends the following :—

R. Liquoris Strychninæ Hydrochloridi	ʒss.
Acidi Hydrochlorici Diluti	ʒiss.
Tincturæ Aurantii	ʒvi.
Aq. Chloroformi	℥vii.

Misce. Ft. Mist.

One-eighth part to be taken twice or thrice a day, with, or after meals.

Or,

R. Liquoris Strychninæ Hydrochloridi	ʒss.
Acidi Phosphorici Diluti	ʒiss.
Tincturæ Quininæ	ʒvi.
Aq. Chloroformi	℥vii.

Misce. Ft. Mist.

One-eighth part to be taken twice daily.

Sir J. W. Moore suggests that it is advisable to interrupt the taking of either of the above mixtures for a few days should they cause headaches or spasmodic pains, due to the accumulation of strychnine.

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Preparations.

OXO AND LEMCO.

(London : 4, Lloyd's Avenue, E.C.)

Oxo is a pure beef beverage, rich in nutriment and pleasant in flavour, and is a strengthening and stimulating liquid food, that embodies in essence the blood-making and flesh-forming properties of the best lean beef.

It is prepared from cattle, bred on farms in the vast cattle-rearing district of the River Plate, where the purest air and water, combined with very fruitful soil, provide an ideal pasturage.

LEMCO is the name by which Liebig's Extract of Beef is now known, and is manufactured by the special process invented by Baron Justus von Liebig. Dissolved in boiling water, it produces strong and clear beef-tea, and it is also of excellent service in the kitchen for the manufacture of soups, sauces, gravies, and entrées ; but, from our point of view, its value in the sick-room and in hospitals can hardly be over-estimated.

Both Oxo and Lemco are invaluable as restoratives in cases of convalescence after such a debilitating disease as influenza. They are both prepared from oxen fed on the best of pastures, and by a process wholly above suspicion.

ARMSTRONG'S BEEF AND MALT WINE.

(James Murray & Sons, Ltd., Glasgow.)

This wine is one of those excellent preparations which are found to be so useful in restoring strength to the weak, and for acting as a tonic to the convalescent. Its beneficial effect in the debilitating sickness after influenza is undoubted, and in conditions of convalescence from other acute diseases, in which patients from various causes are unable to partake of ordinary nourishing and restoring foods, it is not easily surpassed.

It is composed of a well-matured sound port, and contains beef juice and malt extract.

HALL'S WINE.

(Stephen Smith & Co., Ltd., Bow, London, E.)

Hall's wine has been so long before the profession that it needs no fresh introduction now. But many who recommend it, and who have observed the beneficial results following its use, may not know its constituents. In order that they may know exactly what they are recommending, we are enabled to state what the ingredients are. The wine is a compound of the extractive alkaloid of coca leaves, light tawny port wine, and of "Lemco," the modern name given to the Liebig Company's Extract of Beef. The extractive principles of coca leaves is represented by 1 drachm of the leaves to 2 ozs. of port wine. A wineglassful of Hall's wine, therefore, contains the alkaloidal extract of coca leaves amounting to ·15 gr., or about one-seventh of a grain in 2 ozs. of wine and 5 per cent. of Lemco. This is the formula which was adopted twenty years ago, and according to which the wine is prepared to-day. The wine is, therefore, a tonic restorative, and is of great value in all cases of prostration, especially in that succeeding an attack of influenza.

PANOPEPTON.

(Fairchild Bros. & Foster, New York.)

(Burroughs Wellcome & Co., London, E.C.)

Panopepton is essentially a food for invalids, and is prepared from the two great types of food, beef and wheat. In its preparation, the best lean beef is finely minced, and subjected to peptic digestion, until its entire digestible substance is converted into solution, then clarified, sterilised, and reduced *in vacuo*. The finest wheat obtainable is crushed, and submitted to both proteolytic and amylolytic digestion, until its entire nutritive substance is converted into solution, and likewise sterilised, clarified, and concentrated. These two solution are then assayed, mixed in due proportion, concentrated to dryness and dissolved in sound Spanish sherry. The final clarified product is again sterilised. In the preparation, great care is taken in order to preserve the soluble savoury extractives of the beef and whole wheat for their peculiar value as food constituents, and in order that the food may have a characteristic natural wholesome food flavour.

The composition of Panopepton is: proteids 6 per cent., carbohydrates 13 per cent., ash 1 per cent., total solids 20 per cent., the proteids determined in the usual manner by the modified Kjeldahl method, N × 6·25. This constitutes a nuitritive balance of 1 of proteid to 2·16 carbohydrates. Panopepton is a nourishing restorative, stimulating liquid food of great value for the sick in all cases of acute disease, such as influenza and the various infectious fevers, and also for convalescents after exhausting diseases. This and similar foods should, of course, be given only under the guidance of the medical attendant.

PYRENOL (DR. HOROWITZ).

(Goedecke & Co., Leipzig, Germany.)

(Reitmeyer & Co., 93, Crutched Friars, London E.C.)

Pyrenol is a white crystalline powder, readily soluble in water, and has a slightly aromatic odour, a mellow sweetish taste, and is absorbed with extraordinary rapidity. It is also prepared in tablets of 7½ grains each, and the dose varies, according to the exigencies of the case, from 3 to 15 grains, three times a day. It is a valuable remedy in diseases of the respiratory organs, such as asthma, pneumonia, bronchitis, and influenza. In addition to exercising an expectorant and sedative effect, it possesses a very useful antipyretic property, while in chronic, and more especially the putrid, form of bronchitis, its effect as a disinfectant has proved very serviceable. Clinical and physiological researches have also proved it to be non-toxic, and therefore useful where there is cardiac deficiency. Its value in all cases of respiratory complications consequent upon Influenza is therefore considerable. The tablet form of Pyrenol is especially convenient, and small phials, each containing 20, are readily obtainable.

HYDROXYL-FREE COD LIVER OIL.

(London: Peter Moller, 18, High Holborn, W.C.)

The value of Cod Liver Oil needs no amplification from us; its nauseous and objectionable flavour has formed the only objection, and it is gratifying to be able to find a Cod Liver Oil, possessing all the therapeutic qualities of the remedy, which can be taken without causing the usual unpleasant eructations, or "repeatings." These eructations are attributable to the formation of hydroxyls in the oil by the action of the oxygen in the atmosphere, the hydroxyls causing rancidity. Möller's Hydroxyl-free Cod Liver Oil is produced by completely excluding the action of free oxygen from the oil throughout the course of preparation, and from the time the oil leaves the liver cells, until it is bottled, corked and sealed, the whole process is conducted under an atmosphere of carbonic acid. The result of this careful treatment shows a clear, bright oil, which looks quite inviting. We have personally tested, and can confidently recommend it.

THE PRACTITIONER.

FEBRUARY, 1907.

THE TREATMENT OF SOME OF THE FORMS OF GOUT.[1]

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IN this paper, I propose to deal with the treatment of gout in some of the more frequently occurring forms, in so far as such treatment falls within the scope of the practitioner, and is capable of being carried out at the patient's home. Treatment by hydro-therapeutic measures, by climate, by electric light baths, superheated air, etc., will not be dealt with.

Gout is a disease which is due to faulty metabolism, probably both intestinal and hepatic, as the result of which some toxin or toxins are produced and lead to an auto-intoxication, which is an early factor in the development of the gouty condition. This auto-intoxication coincides with, or is followed by, in the majority of cases, a deposition of sodium biurate in certain of the joints or tissues, which constitutes the climax of the gouty attack. For the past few years, my thoughts have been directed, with increasing intensity, to the view that the intestinal tract is a very powerful factor, if not the primary factor, in the development of gout. My attention was first directed to this view, as the result of some clinical observations which I was making in an attempt to explain the action of colchicum in cases of gout. I happened, at that time, to see some cases of acute colchicum poisoning, which, in many respects, resembled acute arsenical poisoning. In toxic doses, colchicum is first a gastro-intestinal irritant ; it produces nausea, vomiting, choleraic diarrhœa, and rice-water stools, just as arsenical poisoning does. It also, in sufficient toxic doses, produces cardiac depression, and a bilateral neuritis, similar to that produced by arsenic. If, then, in therapeutic doses, the main action of colchicum is upon the gastro-intestinal canal, its rapid efficacy, in cases of gout, is possibly due to its effect upon those abnormal intestinal changes which constitute the primary factor in that disease. Colchicum increases the secretion from the intestinal mucous membrane, and so either prevents the formation, or effects the destruction, of the intestinal toxin which, when absorbed into the circulation, is responsible for the development of gout.

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[1] A lecture delivered at the Medical Graduates' College and Polyclinic

URIC ACID.

Not only in the medical world has an unmerited importance been attached to uric acid as a factor in the causation of disease, but unfortunately, among a considerable section of the public, there has arisen a fetichism of uric acid, which has been pandered to, and fostered by, the proprietors of the various quack remedies that are so persistently advertised as being capable of dissolving or removing uric acid from the system. The time has come to clearly recognise that uric acid possesses no toxic properties worth speaking of. The joint manifestations of gout are dependent upon much more general, and much larger conditions than a mere excess of uric acid in the blood. The deposition of sodium biurate is merely the sign of the disease, not the essence of it. In fact the rôle of uric acid in gout may well be compared with that of sugar in diabetes. In support of my belief that uric acid is practically devoid of toxic properties, I would adduce the following facts:—(1) Animals have been made to ingest large quantities of uric acid with their food, and urates in solution have been freely injected into their veins, without eliciting any signs of poisoning: (2) Experiments on frogs' muscles have shown that muscle rigidity and tetanus are produced by hypoxanthin, but that uric acid is inert: (3) After carefully searching the literature on the subject, the only evidence I can find of discomfort following the ingestion of uric acid is the statement of Dr. Walker Hall, who, after taking large doses of uric acid had malaise and headache lasting for several hours. Almost any substance, however (common salt for example), will produce toxic effects, if taken in very excessive quantities.

The contention that a meat diet is poisonous to the human body, on account of the uric acid that it contains, or produces, is preposterous, in view of the facts that many races have maintained robust health on such a diet, and that, for centuries, the beef-eating Englishmen have managed to spread and advance knowledge and civilisation, and to acquire territory in all parts of the world. Surely, if meat is the poison which a certain class of enthusiasts and fanatics maintains it to be, we as a nation should have ceased to exist long ere this. Dr. Harry Campbell, in his interesting series of articles on "The Evolution of Man's Diet," has shown that man has evolved from the ape on a highly animalised diet, and that it was on such a diet that the intellectual faculties, and the faculty of language, which distinguish him from the beast, were developed. It is interesting to note that the recent remarkable advance of the Japanese to the position of a first-class power among the nations is concurrent with the adoption of a more animalised diet by them. On the other hand, the tendency with most people in this country, as I shall have occasion to remark later on, is to eat too much, and to masticate too little, and this applies not only to the consumption of meat, but of all other solid articles of diet.

The treatment of gout should have for its aim the following objects:—(1) The treatment of the gouty paroxysm in cases of acute gout, and the relief of the pain as speedily as possible; (2) the treatment of the subacute, or chronic condition, and the prevention of the recurrence of an attack; (3) the treatment of the affected joint, or joints, with the object of removing the uratic deposits, and of preventing permanent deformity; and (4) the treatment of the various forms of irregular, or abarticular gout.

THE TREATMENT OF ACUTE GOUT.

In order to arrest the abnormal intestinal fermentation, to remove the excessive numbers of intestinal bacteria, and to relieve the catarrh of the intestinal mucosa, all factors in the development of abnormal intestinal toxins, the bowels should be freely opened with four grains of calomel or "blue pill," followed by a saline aperient. For the first twenty-four hours, it is preferable that no food should be taken, but water should be drunk freely.

For the treatment of the gouty paroxysm, the limb should

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be placed in the horizontal position, or slightly elevated above the level of the body, and a cradle should be arranged so as to take the weight of the bed-clothes from the affected part. To alleviate the severe pain felt in the affected joint, warm packs should be arranged round it, consisting of cotton-wool saturated with a soothing lotion, and then lightly covered with oil-silk. I have found the following lotion most useful in relieving the local pain :—

R. Sodii Carb. - - - - ʒiii.
Linim. Belladonnæ - - - - ℥ii.
Tinct. Opii - - - - ℥ii.
Aq. - - - - ad ℥viij.

A small portion of the lotion should be mixed with an equal quantity of hot water, and then poured on cotton-wool previously arranged round the joint. The pack should be changed every four hours. In connection with the acute paroxysm, no attempt at local depletion, such as the application of leeches to the inflamed joint, blistering, or incisions, should on any account be made, owing to the great liability of thereby extending the inflammatory condition, and so producing subsequent ankylosis or deformity.

For the internal treatment of acute gout, colchicum is one of the most valuable drugs that we possess. It should be especially used for acute gout, and for sub-acute attacks supervening on chronic gout. If it is used continuously, tolerance is apt to be acquired, and then the drug ceases to act. At the commencement, a large dose of 30 to 40 minims of colchicum wine should be given, followed by a mixture containing in each dose 15 to 20 minims of the wine, with from 40 to 60 grains of citrate of potassium, which should be administered three times a day. The citrate of potassium, which is given for its combined properties of acting as a diuretic and of diminishing the acidity of the urine, may, if desired, be given as an effervescing mixture, using 30 grains of potassium bicarbonate to 20 grains of citric acid. Colchicum reduces the gouty inflammation, relieves the pain, and shortens the attack. It should only be taken under medical advice, and should never be given in such doses as to produce extreme depression ; after the inflammation of an acute attack has subsided, the doses of colchicum should be gradually diminished until the drug is left off. A very useful method of administering colchicum is in the form of its active principle, colchicine, which may be given in doses of one-fiftieth to one-eightieth of a grain three or four times a day immediately after food. Only a few patients will tolerate doses of one-fiftieth of a grain, the contra-indication of such a dose being the production of diarrhœa and intestinal griping. The following constitutes a very useful pill :—

R.	Colchicinæ - - - -	gr. $\frac{1}{60}$.
	Ext. Nucis Vom. - - -	gr. $\frac{1}{4}$.
	Ext. Hyoscyami - - -	gr. $\frac{1}{2}$.
	Ext. Gentianæ - - - -	gr. i.

After the initial free purgation, as previously mentioned, it is not desirable to produce too free an action of the bowels. All that is necessary is to have a sufficient action to relieve portal congestion and intestinal catarrh. The following pill effects this purpose, in most cases, very well. It is administered at night, and is followed up, when necessary, by a dose of saline in the morning :—

R.	Leptandrin - - - -	gr. i.
	Iridin - - - - -	gr. i.
	Ext. Hyoscyami - - -	gr. i.
	Ext. Colocynth. Co. - - -	gr. iss.

If the pain of an acute attack of gout is so severe as to prevent sleep, seven grains of veronal, or ten grains of trional may be given, or a full dose of extract of hyoscyamus will, in some cases, act as a very useful anodyne. The administration of opium or morphine should, if possible, be avoided owing to the risk of its deficient elimination, and also on account of its diminishing the amount of urine, and its tendency to derange digestion and to check hepatic metabolism.

THE TREATMENT OF SUBACUTE AND CHRONIC GOUT.

In addition to colchicum, which may be given in small doses, guaiacum may very usefully be administered as an alterative which stimulates the metabolism of the liver, and also affords relief to the portal system. From 5 to 10 grains of guaiacum resin should be given in cachets two or three times a day, according to the effect on the bowels, since guaiacum sometimes acts as a laxative. The method of administering the powdered guaiacum resin in cachets is far preferable to giving the tincture of guaiacum in a mixture, as, in the latter form, a nauseous medicine is produced, and the precipitated resin tends to cling obstinately to the tongue and fauces. In cases of chronic gout, the colchicum may be very conveniently administered in the form of the colchicine pill, given three times a day. In order to encourage the elimination by the kidneys of the toxic agents of gout, citrate or bicarbonate of potassium should be employed as a diuretic, which increases the volume of the urine, and, at the same time, diminishes its acidity. The use of the potassium-salt may with advantage be pushed until moderate alkalinity of the urine is produced, as, by such means, the tendency to the deposition of uric acid, or sodium biurate, in the kidney-tissues is removed. Free diuresis should also be encouraged by the drinking of sufficient quantities of water. As to the beneficial effects of employing a potassium-salt in conjunction with colchicum in the treatment of acute and subacute gout, I am fully assured, and my experience is that, of the various potassium-salts, the citrate is the most useful. If given in sufficiently large doses, it tends, by its conversion in the kidneys into the carbonate, to diminish the acidity of the urine, which is generally high in connection with the gouty paroxysm, while, at the same time, it increases the solvent power of the urine for the uric-acid salts, and so assists their elimination. In cases of sluggish action of the liver, of gastro-intestinal catarrh and torpor, of gouty dyspepsia, and of other forms of irregular gout, in which there are no appreciable uratic deposits in the joints, mineral waters containing sodium-salts are undoubtedly beneficial, owing to the action of those salts as hepatic and gastro-intestinal stimulants.

As regards the use of lithium-salts in the treatment of gout, my opinion is that they are not so useful as the potassium- and sodium-salts. The principal objection to their use is their greater toxicity, and depressing action on the heart, as compared with the potassium-salts. They consequently have to be given in such small doses that I am very doubtful as to whether, in such doses, they possess any remedial effect at all. On the other hand, I constantly meet with patients suffering from cardiac depression, and even dilatation, as the result of the excessive and continued consumption of lithiatablets, which are so persistently, so speciously, and so wrongly vaunted as curative of gout.

The enlargement and tenderness of the gouty joints is due to two causes, the deposition of sodium biurate in the cartilages and fibrous structures, and a chronic inflammatory thickening of the fibrous tissues. For the reduction of this thickening, as well as for painful gout of the sole of the foot, and for gouty neuralgic affections, iodide of potassium, given internally, is a useful remedy. In cases of gout, associated with the contracted granular kidney, as evidenced by slight albuminuria and high arterial tension, the administration of iodide of potassium is also most beneficial. I usually prescribe it in doses of 10 grains three times a day, and continue its use over a period of six or eight weeks. My experience is that it seems to act more beneficially when given in combination with the compound decoction of sarsaparilla.

THE LOCAL TREATMENT OF GOUTY JOINTS.

If much swelling of a joint persists, the limb should be elevated as much as possible, and a light flannel bandage applied to the joint. If the œdema persists, the hot douche, followed by sponging with a cold strong solution of common salt, will be found serviceable. The application of the so-called solvents of uric externally to affected joints is useless, as they are not solvents of sodium biurate. Neither is there a possibility of converting the uric acid in the deposits into a more soluble condition by the administration of potassium or lithium salts, or by means of any of the so-called solvents of uric acid. The Scotch douche is very useful in the treatment of chronically affected joints. A good-sized stream is thrown with considerable force upon the affected joint, employing first cold water for half a minute and then hot, the latter should be as hot as the patient can bear, and should be continued for one minute. This process is repeated for fifteen or twenty minutes. The repeated alternations of temperature produce a stimulating effect upon the circulation about the joint, and so increase tissue change, and favour absorption. Massage of the joint should be resorted to immediately after the douching, as the tissues are then in a relaxed condition. In many cases of chronic articular gout, the salt pack is efficacious. It consists of flannel soaked in a warm saturated solution of common salt, which is wrapped round the affected joint, covered with oiled silk and a bandage, and kept on all night. It should be repeated nightly as necessary.

For the removal of œdema, associated with considerable uratic deposits in the joints, there is nothing better than the application of galvanism to each of the affected joints for a few minutes, using a current of about 10 milliampères, with the negative pole over the affected region. The galvanism should be followed by a few minutes' massage, to hasten absorption, and to quicken the circulation. Careful rubbing with iodide of potassium and soap liniment may also be employed to relieve the stiffness and thickening around joints.

THE TREATMENT OF IRREGULAR GOUT.

Gouty Dyspepsia and Acidity.—In addition to the usual remedies, such as bismuth subcarbonate, sodium bicarbonate, bitters, etc., taka-diastase is a most useful drug in the treatment of gouty dyspepsia. It is made up in the form of tablets containing two and a half grains in each tablet, and one of these should be taken immediately before each meal. The taka-diastase encourages the digestion of the carbohydrate elements of the food, and so prevents the development of fatty acids, which, by their irritating effects, are so common a factor in the development of gouty dyspepsia.

Hyperchlorhydria, or excessive secretion of hydrochloric acid in the gastric juice, occurs in some gouty subjects, and in such should be regarded as a form of irregular gout. By this I do not mean that hyperchlorhydria occurs only in gouty subjects, as I have met with many cases of it in individuals who have no tendency to gout, either inherited or acquired, but I have on several occasions met with it in gouty people, and have not uncommonly seen it alternate, either with attacks of regular gout, or of some other form of irregular gout. The gastric discomfort does not begin until from one to two and a half hours after a meal, and generally continues up to the next meal, which for a time relieves it. The discomfort may simply be one of distension or oppression, or may amount to severe pain, which is generally described as being of a burning character. The pain is usually referred to the epigastrium, but may also be felt beneath either shoulder blade. The condition is usually accompanied by flatulent distension, acid eructations, and severe heartburn. The eructations give, for the time, considerable relief as a rule. The effect of this excessive secretion of hydrochloric acid is to cause a too rapid digestion of the proteid elements of the food, with retention in the stomach of the starchy and fatty constituents of the food. In consequence of this retention the gastric secretion is kept up, while, at the same time, no constituent is present with which the gastric juice can combine, and, in consequence, an excessive amount of free hydrochloric acid accumulates in the stomach. Undoubtedly some gouty subjects have the habit of secreting an excessive amount of gastric juice, and especially does this occur as a consequence of the consumption of a diet too rich in nitrogenous substances.

The treatment of this condition consists in a proper regulation of the diet, by cutting off any excess of the proteid articles of diet, and by neutralising the superfluous acid by the administration of some alkali. A drug that I have found most useful in the treatment of this hyperchlorhydria is hopogan (the magnesium peroxide). It not only gives immediate relief of the pain and discomfort, by its neutralising effect on the excess of acid, but it also parts with one-half of its oxygen, and acts as an internal antiseptic. It is a most valuable drug in many abnormal gastric and intestinal fermentations. It is a white tasteless powder, and is best given in a little milk in doses of 20 to 30 grains three or four times a day, taken one hour after meals. If it exerts too great a purgative effect, the dose should be diminished. It is also very useful in allaying the irritation, in many cases, of gouty pruritus, which is probably due to absorption of a toxin or toxins from the intestinal tract. In cases of ordinary neurotic dyspepsia, associated with flatulence, the drug is, in my experience, of no value whatever.

Hepatic Torpor.—A very common form of irregular gout is due to defective metabolism of the liver, and is known as hepatic torpor, or hepatic inadequacy. In this form, the fæces are pale, generally very offensive, and, as a rule, constipation occurs. Slight jaundice is usually present, as evidenced by a yellowish conjunctiva and muddy complexion, and the urine is high coloured, of high specific gravity, and very acid. In the treatment of this form of irregular gout, the most important consideration is the restoration of the liver to its normal state of activity, and here the alkaline sodium-salts are especially useful. There is no better treatment at the outset than a dose of "blue pill" or calomel at night, followed by a dose of Epsom salts or Carlsbad salts in the morning. Subsequently, a pill containing a small dose of "blue pill" or calomel, combined with euonymin and colocynth, will be found most useful. In such cases of gouty hepatic inadequacy, a mixture, which I have found most beneficial as regards its stimulating effect on the metabolism of the liver, and also of the gastro-intestinal tract, is the following, which should be taken a quarter of an hour before meals :—

℞	Sodæ Bicarb. - - - - -	grs. xii.
	Tinct. Nucis. Vom. - - -	♏x.
	Tinct. Gentian. Co. - - - -	ʒss.
	Sp. Chloroformi - - -	♏xii.
	Aq. Menth. Pip. - - - ad	℥j.

Gouty Eczema.—Of the purely internal causes of eczema, disorder of the gastro-intestinal tract should, in my opinion, be placed first. The gouty person is one, who is not only liable to such disorder and to faulty assimilation of food, but is also particularly vulnerable to inflammations of the synovial and mucous membranes, and of the skin. Some authorities have doubted whether there is such a disease as a gouty eczema pure and simple, but it is a common experience, in my practice, to find that eczema is very prone to occur among the gouty. Among the descendants of gouty ancestors, I frequently find that certain members suffer from recurring attacks of eczema, while others suffer from regular gout. Again, with some individuals, it is not uncommon to find attacks of eczema alternating with attacks of articular gout, or to find that an attack of eczema subsides with almost startling rapidity when an attack of true gout supervenes, or *vice versa*. It is true, that it is impossible to diagnose a gouty eczema at sight, but a careful inquiry into the patient's history, and into the family history, makes the diagnosis, in my opinion, in many cases a certainty.

In the treatment of this form of irregular gout, special attention should be given to two details. One is to see that the bowels are freely opened, which, at the outset, may be secured by the administration of "blue pill" or calomel, followed by a saline; the other point is, that entire abstention from alcohol is most desirable, at all events during the treatment and persistence of the eczema. It is best that any form of alcohol should be abstained from, but the prohibition applies more especially, in my experience, to the red wines. I have met with several cases, occurring among gouty individuals past the middle age of life, in whom two or three glasses of claret or burgundy will, in the course of a few hours, cause the development of an eczema. During the irritative stage of dry gouty eczema, I have found the application of a lotion, consisting of liquor plumbi subacetatis ʒj, liquor carbonis detergens ʒj, aqua sambuci ad Oj, most soothing, especially if followed by the use of a simple dusting-powder, such as cimolite powder. For the acute moist type of eczema, a similar lotion, but with a preparation of opium replacing the tar solution, is advisable. In cases of gouty pruritus, or, as it is sometimes termed, latent gouty eczema, the severe itching is frequently relieved by the use of carbolic-acid lotion. When the eczema is in a chronic condition, much benefit is usually experienced from immersion in sulphur-baths. After the bath the skin should be carefully dried, and a dusting-powder such as cimolite powder freely applied. In cases of gouty eczema and gouty pruritus, a careful dietary must be enforced, care being taken to forbid all articles which the experience of the patient in the past has shown to produce dyspepsia. Persons who are subject to attacks of gouty eczema should avoid such acid fruits as strawberries, gooseberries, apples, and pineapples; rhubarb also should be excluded from the dietary. As regards the medicinal treatment of gouty eczema, my experience is that it is not necessary to give the ordinary anti-gout remedies, such as colchicum, etc. It is much more important to treat the dyspepsia and the catarrhal condition of the gastro-intestinal tract, which are generally present, as associated or causative conditions, by the administration of subcarbonate of bismuth with the bicarbonate of sodium, or the bicarbonate of potassium.

Insomnia in Gouty Subjects.—Many gouty persons complain of insomnia or restlessness when in bed. This is frequently either toxic in its origin, or is due to a state of high tension in the cerebral arteries. As a rule, the insomnia is not complete, but consists of restlessness, interspersed with varying intervals of light or broken slumber. In such cases, careful attention should be given to the state of the pulse, the heart, and the condition of the urine. In many cases it will be found that the pulse is of high tension, and is associated with accentuation of the aortic second sound, perhaps reduplication of the first sound in the vicinity of the apex of the heart, and a slight degree of albuminuria. In such cases the insomnia is best relieved by the administration at night of small doses of "blue pill" or calomel, combined with full doses of the extract of hyoscyamus. Bromide of ammonium may also be given as a sedative, and as a drug which reduces arterial tension, but with such patients it is most undesirable to resort to the use of the ordinary hypnotic drugs.

It will perhaps be noticed that I have made no reference to the employment of any of the numerous preparations, which are so constantly being introduced to our notice as infallible solvents of uric acid, and as specifics for gout. It is not that I have not made trial of them, but such trials have always led to disappointment, and have sent me back with renewed confidence to the drugs of our Pharmacopœia. I have witnessed, and doubtless shall again witness, the rise and fall in popularity of many a so-called solvent of uric acid, which has been introduced with such a flourish of trumpets, and with the usual undeserved laudation born of insufficient experience.

DIETETIC TREATMENT.

It is not my intention to do more than refer to some of he general principles underlying the dietetic treatment of gout. In gout, the chief source of infection is the alimentary tract, and an injudicious diet acts mainly by its influence on the bacteria present in the digestive tract. The growth of bacteria in the intestinal tract varies with the amount of proteid food, and the degree of catarrh present. The greater the amount of proteid food taken the greater will be the number of intestinal bacteria present, and an increase in the catarrh of the intestinal mucosa not only increases the number of bacteria, but also renders them more pathogenic in character. These facts to a great extent explain why it is that excess of proteid food, and especially of those forms of proteid food liable to bacterial changes, and of certain wines and beers, which tend to the production of intestinal catarrh, exercise so strong an influence in the production of gout. It is essential that there should be an accurate adjustment of the food to the capacities of the intestinal mucosa and of the liver, for many of the symptoms of gout, and especially of irregular gout, are due to hepatic inadequacy. Purin containing foods should not be entirely withheld, but should be given in such amounts as the individual can properly metabolise.

I have for many years held, and taught, the opinion that the great majority of people take an excessive amount of proteid food. The comparatively recent work of Chittenden indicates that the hitherto prevailing ideas as to the quantity of proteid food necessary for the maintenance of vigorous health and strength err on the side of great excess, and that a diet, both poorer in proteid and lower in total fuel value, is the rational one. There can be little doubt that the reason why so many apparently conflicting dietary systems claim, and to some extent successfully claim, good results, is due to the reduction in the quantity of food, and especially of the proteid food, rather than to the alteration in the quality of the food.

An ideal diet is one which will supply the true needs of the body, which will maintain the highest degree of physical and mental activity, and which will not over disturb the nitrogen equilibrium. If the nitrogen output persistently exceeds the nitrogen intake, then the body is feeding on its own tissues, and, therefore, the proteid of the food is insufficient in amount. Chittenden's experiments show that it is quite possible to maintain body weight, and keep up nitrogen equilibrium, and preserve strength, vigour, and good health on from 34 grams to 56 grams of proteid matter per day. My own experience is that, on a diet containing this amount of proteid, gouty persons maintain their nitrogen equilibrium, and body weight, become free from most of their gouty symptoms, and generally enjoy a good condition of health.

Animal Food.—It must be remembered, on the one hand, that animal foods constitute to the majority of people the most attractive and appetizing forms of diet, and are, therefore, likely to be taken in excess; hence the necessity for limiting the amount to be taken. On the other hand, it is desirable to increase the combustion and the oxidative powers within the tissues. In my opinion, it is absolutely erroneous to exclude from the dietary of the gouty, such articles as meat, fish, and tea, because they are assumed to contain uric acid. No class of food-stuff gives so great an amount of energy, and produces so much heat as animal food, and no class is more easily digested by the majority of gouty people. On the whole, it may be stated, that animal food, such as fish, chicken, game, and meat, is best suited to the majority of cases, whilst foods of the farinaceous class are most likely to disagree. White meats, such as chicken and fish, are more digestible than red meats. The quantity of meat, and especially of red meat, must be restricted in those cases, in which the kidneys are imperfectly performing their eliminatory functions, as evidenced by a pale urine, of low specific gravity, deficient in urea and purin bases.

Vegetable Foods.—A fair proportion should be taken with two meals each day. The choice of vegetables will depend upon the digestive capacity of the patient; but, excepting the potato, as a rule, those that grow above ground are preferable to root vegetables. At the same time, it must be borne in mind that, with certain patients some of these vegetables may produce some form of dyspepsia, and I cannot too strongly urge that, in the dieting of the gouty, no hard-and-fast rules can be laid down, but the idiosyncrasy of each patient to various articles of food must be made the subject of careful observation and study. Due consideration should also be given to the patient's experience of what articles of diet disagree and agree with him.

Simplicity of Meals.—The diet should be simple, that is, the meals should not be made up of too many articles. Simplicity of food means facility of digestion. Moderation, in both eating and drinking, is perhaps one of the most essential points to insist on. Certainly meat, even red meat, should not be excluded. Those articles of diet, that are known in the individual to favour intestinal fermentation and putrefaction, should certainly be avoided, and, speaking generally, a sense of discomfort after a meal indicates that some food has been taken which is not beneficial to the individual in his present condition. If the gouty symptoms are due to over-production of toxic material from faulty intestinal and hepatic metabolism, and if, at the same time, the kidneys are sound, then a diet which mainly consists of animal food is indicated, and in extreme cases of this class even the so called "Salisbury Diet" may be useful. If, on the other hand, the symptoms are due to defective elimination on account of diseased kidneys, then a more vegetarian diet will be best. The value of the so-called "Salisbury Diet" consists in the small amount of energy necessary for the digestion of so simple a diet, and, in the fact, that it contains little which can set up intestinal fermentation or putrefaction. On the other hand, a strictly vegetarian diet requires more digestive energy than a purely animal one, and a much larger quantity of vegetable food must be taken to produce an equal nutritive effect. If, during the treatment of gout, an attack of gouty dyspepsia should intervene, a milk diet should be employed until the dyspeptic symptoms have abated.

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POSTERIOR TARSAL RESECTION OF THE FOOT BY THE METHOD OF MIKULICZ AND WLADIMIROFF,

WITH REPORT OF A CASE, AND REMARKS ON THE OPERATIVE TECHNIQUE, AND THE APPLICABILITY OF THE PROCEDURE.

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[With Plates I. and II.]

When, in 1881, the late Professor von Mikulicz-Radecki published an account of the operation of posterior osteoplastic resection of the foot,[1] which is now generally known by the associated names of himself and Wladimiroff, he laid down, as the possible indications for its adoption, the following three conditions:—

I. Caries of the foot, where the disease is confined to the astragalus, the os calcis, and the ankle joint;

II. Extensive loss of skin substance in the region of the heel;

III. Injuries, especially gunshot, with destruction of the heel.

His own first published case belonged to the second of these classes, being one in which the region of the heel was the seat of extensive syphilitic ulceration, and the operation was probably fully justified, not only by the satisfactory functional result, but by the fact that the syphilitic nature of the ulcerative process would have offered a pretty hopeless bar to any satisfactory procedure of a more simple plastic type.

It may, however, be questioned whether, in the light of more recent knowledge and perfected surgical technique, the adoption of any definitely maiming operation, as the Mikulicz-Wladimiroff resection certainly is, can be regarded as justified in any condition of a simple nature, such as would be included in Class II. of Mikulicz.

Mere loss of skin substance, however extensive, and though involving a region exposed to pressure like the heel, would not now warrant so heroic a remedy as this operation implies. Plastic surgery has recently made so substantial an advance that even the most extensive loss of skin substance may be readily made good by grafting, especially by means of the method originally proposed by Wolfe of Glasgow,[2] and further perfected by Krause and others.

In the light of my own now fairly extensive experience of the "whole-skin" method of skin-grafting,[3] first of all in making good extensive defects following severe burns, and, more recently, in covering a variety of skin defects, where, for anatomical and other reasons, the prospects of success might have been regarded as much less hopeful, I have come to realise very clearly that the cases must indeed be few in which any severe maiming operative procedure could be justifiably employed, and that such a procedure could very rarely be allowed to pass muster, unless a full and efficient trial had first been made of these simpler methods without success. For this reason it seems necessary to question the present justification of Mikulicz's second class.

Class III., which embraces cases in which, along with the damage to the soft tissues, extensive injury to and loss of bone has occurred, may be allowed to stand, though it is unlikely that there will be found, even in this category, any considerable number of cases.

Class I. has furnished, up to the present, the greater portion of all the cases submitted to the operation, and in all probability it will continue to do so.

Since the appearance of Mikulicz's paper there have been advanced by different surgeons some further applications of the procedure he described.

Thus at least three cases of malignant growths in the region of the heel have been submitted to this method of treatment, Sklifassowsky adopting it, in 1885, in a case of osteo-sarcoma of the os calcis, and, in 1886, in a case of periosteal sarcoma of the same bone, in both, apparently, with satisfactory result.

In the same year as the former of Sklifassowsky's cases, Wahl employed the method in a case of melanotic sarcoma of the skin of the heel, and with a good result.

Another application of the operation has been found in adding to the length of a limb unduly shortened by accident or disease. Two cases, reported respectively by Caselli, in 1885, and by Rydygier, in 1890, illustrate this. Caselli made good a large part of the shortening (7·5 cm.), following upon a spontaneous dislocation of the hip, while Rydygier, in a case in which the knee-joint had been resected on account of tuberculous disease, and in which there resulted failure of growth, with consequent considerable shortening, was able to lengthen the limb to the extent of fully 5 cm., and thereby to enable the patient (a youth) to recover the power of walking.

In several recorded cases of suppuration in the ankle and tarsal articulations, this operation has proved useful, as, for example, in a case reported by Gellé, in 1891, in which suppuration of the ankle joint was associated with a severe fracture of that region; also in a case described by Coëlho, in 1888, in which chronic suppuration and fistulæ about the ankle followed upon a complicated fracture, the typical resection being carried out seven months after the injury.

In a case of chronic osteo-myelitis following injury, in which there was considerable fistulous development and ankylosis of the ankle, Fenger, in 1887, performed this operation with success.

Yet another application for the operation was found by Professor Bruns, who, in 1886, employed it in a case of paralytic talipes, and, in 1887, in a case of severe talipes equino-varus of long standing. In both the result proved satisfactory.

The most complete collection of cases, in which the adoption of this operative procedure has been reported, is that of Kohlhaas, published in *Bruns's Beiträge* in 1891.[4] Kummer, in a paper published a short time before, had been able to collect only 54 references in the literature of the subject.[5] Kohlhaas tabulated, in his collection, 73 cases, and gave a short summary of each in an appendix to his paper.

Of his 73 cases, 51 come into Class I. of Mikulicz, being cases of tuberculous disease of the ankle, and of the posterior tarsal bones.

Classes II. and III. combined account for 12 more, these including also the cases of chronic suppuration, fistulous development, ankylosis, etc.

The balance of the 73 is made up of the few cases already referred to, in which the operation was performed for malignant disease, to lengthen a shortened limb, for the treatment of talipes, and (in three instances) for reasons which are not given.

The list may be tabulated, therefore, as follows:—

Caries of tarsus, etc. (Class I. of Mikulicz) -	51
Loss of substance—skin and (or) bone (Classes II. and III. of Mikulicz), with or without suppuration, fistulæ, ankylosis, etc. - -	12
Malignant disease of heel or tarsus - -	3
Shortening of limb - - - - -	2
Talipes - - - - - - -	2
Unstated - - - - - -	3
	73

Since the date of publication of Kohlhaas's figures, there has probably been a considerable number of further instances of the employment of the operation, but his list sufficiently indicates the large predominance in the application of the method for the treatment of tuberculosis of the tarsus. It may very reasonably be assumed that this will continue to be its chief sphere of usefulness.

It is mainly with the object of illustrating, by one more example, the importance of its scope in this field, as well as to draw attention to certain details in the operation itself, that this further case is now recorded.

E. M., aged 14 years, was admitted to the Western Infirmary, Glasgow, on the 8th November, 1902, suffering from advanced tuberculous disease of the right tarsal bones. The whole foot was swollen and painful, and sinuses on each side led into the ankle joint proper, as well as into communicating channels and cavities throughout the tarsus.

The advanced state of disease, and more especially the mixed infection present, were considered so unpromising that the boy was refused admission to the ordinary surgical wards, and was sent into the ward reserved for septic cases, at that time under my charge. Although there was extensive involvement of the tarsus, and considerable disease of the lower ends of the tibia and fibula, the anterior part of the foot appeared healthy. It may be mentioned that the metatarsal-phalangeal joint of the left great toe was also diseased, but, during residence in hospital, this was apparently quite recovered from.

The condition of the right foot was such, that, short of amputation, it appeared unlikely that any operative procedure, stopping short of the most radical resection of the tarsus, would afford any reasonable prospect of success.

The operation of Mikulicz and Wladimiroff was resolved on.

Operation.—On the 14th of November (six days after admission), a radical resection was carried out, following generally the lines of the procedure mentioned. The skin incisions did not accurately follow the usual or recognised plan, some variation being rendered necessary by the exigencies of the case, particularly with a view to the avoidance of disorganised skin, and actively diseased soft tissues. Of the later stages of the operation the same may be said, for instead of removing merely the os calcis, the astragalus, and the articular surfaces of the scaphoid, cuboid, and tibia, with the malleoli, it was found necessary to take away the whole scaphoid, a good part of the internal cuneiform, practically the whole of the other two cuneiforms, and nearly the whole cuboid, also much more of the tibia than the mere articular surface. The effects of this more extensive removal of bone will be alluded to later.

In the first instance the bones were, indeed, severed at the usual level, but, as the disease could not have been eradicated by this means, the more radical resection was straightway decided on. It was carried out chiefly by bone forceps and gouge. When, at the last, the fore part of the foot was placed in line with the leg, there intervened between the metatarsals and the cut ends of the tibia and fibula only a small section of the internal cuneiform, little more than the distal cartilaginous surfaces of the other two cuneiforms, and the merest shaving of the cuboid.

The result of this extensive removal of bone was seen, to some extent, in the great redundancy of the soft tissues forming the anterior bridge, when the bone surfaces were fixed together; this initial, considerable, anterior bulging gave no trouble, however, and soon in a large measure subsided.

The bone surfaces were fixed in proper relation by sutures of silver wire penetrating the soft tissues as well as the bone.

The subsequent progress was practically uneventful.

In spite of the pre-operation sepsis, healing took place by first intention. The first dressing was not disturbed for some days, and, at the end of a fortnight, the stump was fixed in a plaster of Paris sheath, with the toes in the position of extreme dorsal flexion. This plaster casing was removed, and re-applied, once or twice in the course of the following month, and, in the early part of January, the patient was sent to the convalescent home still wearing a light plaster support, the toes, however, being no longer fixed. These had, by this time, satisfactorily acquired the position of dorsal flexion which, indeed, had come to be their position of rest. Bony union was not yet fully consolidated. A few weeks later, however, when the patient was re-admitted to hospital for a short period, in order to have a suitable artificial foot made for him, bony union had been completely established. This was verified by X-ray photographs, which also displayed the extent of the bony resection.

At the same time, a cast was made of the stump, and photographs of this, as also of the stump itself, were obtained. They are shown in the plates accompanying this paper. (Figs. 1 and 2.)

It may at once be said that the after-history of the case, up to a period of three years from the operation, when the last opportunity offered itself to observe the condition of the leg and stump, has been almost completely satisfactory. A little residual tuberculosis in the lower part of the leg, close to the lower end of the tibia, appeared in 1904, but was readily overcome by simple measures, and, when last seen, in December 1905, the patient was well, and able to undertake quite arduous employment.

The operative treatment may, therefore, be regarded as fully justified by the efficient functional result.

Before referring to the artificial appliance fitted to the stump, on the satisfactory nature of which this result probably so largely depended, it may be permitted to draw attention shortly to certain points suggested by the operation itself, the details of its execution, and the after-treatment.

First, it may be stated that the prospects of effective vitalisation of the stump were never in doubt. It was apparent, during the operation, that the vascular supply, along the anterior bridge of soft tissues, was entirely adequate, for free bleeding took place from the severed terminal branches of the digital arteries on the plantar aspect of the metatarsal section of the stump, immediately on relaxation of the tourniquet, which had been applied to the thigh at the beginning of the operation.

It seems unlikely, therefore, that any modification of the incision so "as to conserve the soft parts on the inner side of the foot, with the vessels and nerves" (quoted from one of the most recent German handbooks[6]) will be found necessary or advisable.

The preservation of the nerves does not seem to be of any greater practical importance than does that of the plantar vessels, though fear of a neuro-paralytic ulcerative process led Roser, in 1884, to carry out suture of the posterior tibial nerve. No justification of such a fear seems, so far, to have come to light in the experience of reported cases

The risk of gangrene following the operation, whether in gross form from vascular defect, or in less extensive degree from neuro-paralytic cause, seems indeed to be quite negligible.

In only one case within my knowledge did gangrene occur. The case in question is referred to by Kohlhaas in his paper in *Bruns's Beiträge*. The patient was operated on by Sordina, in 1885, for tarsal caries. Gangrene developed, and, three days after the typical resection, amputation had to be carried out.

Secondly, reference may be made to the effect of the removal of (1) less, and (2) more bone than was suggested in the original paper of Mikulicz.

The result of performing the typical operation is, necessarily, the production of a certain degree of lengthening of the limb, to the extent, perhaps, of 1·5 cm., and this may require to be compensated for by the fitting of a sufficiently thick cork sole to the boot on the sound side, so as to equalise the two limbs.

In a young subject, such a degree of lengthening need give little cause for regret, especially if the lower epiphyseal line of the tibia has been actually, or even indirectly, affected by the operation, for, in course of time, this lengthening will, in all probability be very largely obliterated by retarded growth of the leg. Conceivably, therefore, a case might occur, in which even less bone than Mikulicz prescribed might be cut away, and the greater length of the limb would be, in course of time, reduced to a satisfactory level in accordance with the inequality in growth of the sound and the affected limbs.

Where, for other reasons, as in the cases of Caselli and Rydygier, a substantial addition to the length of the limb seems desirable, the less bone removed the better.

In the case which I have reported, the condition was quite the reverse of what has just been considered, more bone being removed than the typical operation of Mikulicz demands. The cuboid and scaphoid were not merely cut through close to their proximal articular surfaces, but the former was removed almost entirely, only its distal articular portion being left, while the latter was completely removed.

The cuneiforms were taken away, all but the merest shaving of their metatarsal extremities, while the removal of bone had to be made equally extensive above also, on account of the extent of disease in the lower end of the tibia.

In spite of this extensive removal of bone, the limb proved distinctly longer than the sound one, but the difference was so slight as to be very readily made good by the slightest thickening of the sole of the boot on the sound foot, especially as the artificial appliance worn on the stump, was fitted in a boot having a comparatively thin and hollowed-out sole ; the appliance was also so arranged as to bring the plantar surface of the balls of the toes into as close relation as possible with the sole.

A further point, arising out of a consideration of the case, is as to the prospects of attaining firm bony union. As mentioned in the detailed report, this had not been attained when the patient was dismissed six weeks after the operation, but, when he was seen several weeks later, it appeared to be completely established. This was confirmed by radiograph, and when the patient was last seen, the stump was as firm as ever, and not the slightest degree of bending had taken place at the site of union.

In the recorded cases, the attainment of good bony union has been pretty general, but in two cases, reported by Helferich, in 1887, it was much delayed, and in one of these a secondary operation was performed, the ends of the bones being refreshed with a view to the encouragement of fresh growth.

In one case, reported by Rose, in 1886, union by bone had not been obtained even after a period of six months, and amputation was carried out. It seems at least questionable how far this was justified, for it is well known that, in an ordinary simple fracture, union may be long delayed, yet in the end prove quite satisfactory and complete.

It is probable that any delay in bony consolidation will depend on local or general nutritive defect. Recurrence of local disease, as well as any general depression of tissue vitality, are bound to interfere with bony consolidation, just as in the repair of ordinary fractures.

Further, it seems reasonable to suppose that the more intimate the approximation of the bone surfaces, the less chance there will be of any delay or failure in bony union. The employment of artificial means of approximation and fixation must therefore be regarded as not only justifiable but advisable.

In the description of the operation in the German handbook already referred to,[6] it is suggested that deep catgut stitches through the soft parts should generally suffice. Bone sutures and pegs have, however, been largely used, and it seems probable that they will continue to be employed. In my case sutures of silver wire were passed through bone, soft tissue, and skin, and were removed after a few weeks.

In the detailed description of the case, attention was directed to the early and careful dorsal flexion of the toes. The attainment of good dorsal flexion should be, in my opinion, not the only object aimed at. It seems desirable, as indeed it is, in my view, quite practicable, to obtain a certain degree of mobility of the toes from their strong dorsally-flexed position of rest. The cutting of the plantar flexor tendons, advised by some writers, is not at all necessary to the

PLATE I.

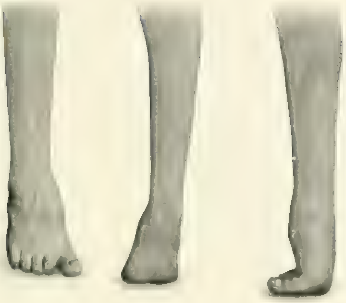
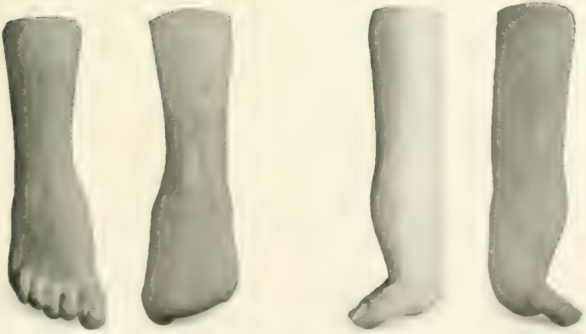


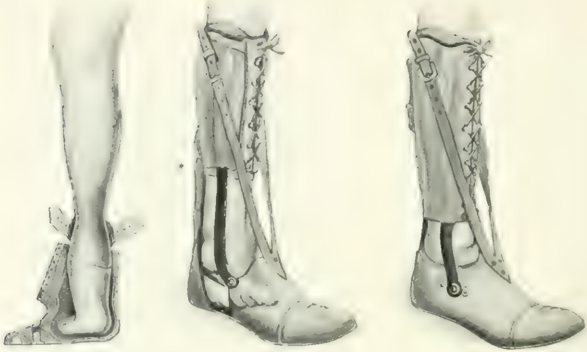
Fig. 1. *Actual stump, showing dorsal flexion of toes and slight posterior scar.*



Front. Back. Inner aspect. Outer aspect.

Fig. 2. *Plaster cast of stump.*

PLATE II.



A. B. C.

Fig. 3. A. *Reproduced from the original plate accompanying Mikulicz's article, showing the resulting stump, dorsally flexed toes, and section of the special boot.*

B. (*Sketched by Dr. John Lindsay*), *to show the appliance used in my case, the movable joint at level of normal ankle movement, the freedom of movement of the toes, and the more efficient support to the leg and stump.* (Note :—*From the point marked by an asterisk downwards, the casing has been cut away in the sketch, in order to show the stump in position.*)

C. (*Also sketched by Dr. John Lindsay*), *to show for purposes of comparison a common type of artificial appliance used after a Syme amputation.*

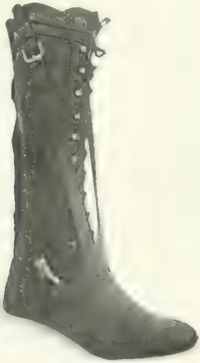


Fig. 4. *Showing the actual artificial appliance used.*

attainment of a rest position in full dorsal flexion, and it will certainly interfere materially with the preservation or acquirement of that degree of mobility of the toes which I regard as a valuable acquisition in the resulting stump. It may reasonably be expected that the intrinsic muscles of the flexor aspect of the foot will have some functional value in the stump, as, indeed, in my case, I am satisfied that they actually had, and the conservation of that function contributes to the maintenance of a degree of elasticity or spring, which is of substantial value to the patient in walking, whatever form of foot gear be employed.

As will be seen in the accompanying illustrations (Fig. 3, B, and Fig. 4), provision was made, in the case I now report, for taking full advantage of this, by leaving the toes free to move in the artificial appliance I had made, and by having a hinge joint so placed on each side as to profit fully from the free play of the toes. In the simpler boot suggested by Mikulicz, a copy of which (Fig. 3, A) is here reproduced from the plate accompanying his original paper, the only provision made for movement is the insertion of a spring joint mid-way between the toes and the tip of the cork-filled anterior portion of the boot. This gives, no doubt, a certain amount of mobility, but, so far as toe movements are concerned, it would be of no value whatever.

Further, it will be obvious from the same illustration that the Mikulicz boot can give no effective support to the leg, or to more than the actual region of the operation scar. Where any doubt at all exists as to the nature of the bony consolidation, the employment of so ill-supported a boot, at any rate during the earlier months, is accompanied by definite risk.

It seemed to me to be wiser to give the long crural lever a much more substantial support, and accordingly the apparatus was made to fit the leg closely and firmly almost as far as the knee. [For comparison, there is reproduced in the same plate (Fig. 3, C.) an illustration of the artificial appliance commonly employed after the typical Syme amputation.]

The use of so apparently elaborate a support, as I have described, may be justified also on other grounds. It is generally recognised that, after a typical resection of the knee, though bony union may at first seem firm enough, it is well to have applied for many months a really effective support, in order to the prevention of gradual bending at the level of union. Such bending is very apt to occur, and at times it does so, even after a long period has elapsed from the date of the resection. A similar bending of bone, apparently firmly consolidated, may very well take place after the Mikulicz-Wladimiroff resection, and the use of a strong supporting appliance is, therefore, well justified.

There is a further and important consideration in justification of its employment, viz., to lessen the risk of strain or injury, however slight, to a part previously the seat of tuberculous disease. Such strain or slight injury may at any moment determine the lighting up afresh of local disease, and anything likely to prevent this must be regarded as justified.

The operation has yielded, then, in the case which I have recorded here, a most satisfactory result. The stump is firm and stable, and, in virtue of the mobile toes, the patient is able to walk, using the appliance I have described and figured, so well, that for one, not cognizant of the actual state of affairs, it is practically impossible to detect any limp or defect in gait. By the operation, the patient regained the power of carrying on his ordinary occupation, and, when I last saw him, was in regular employment as a miner.

Kohlhaas pointed out in his paper[4] that, out of the 73 cases he was able to collect, the patients were able, in 56 of them, to walk "well" or "very well" afterwards. That, surely, is sufficient to place the operative procedure beyond all possibility of question.

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VALVULAR DISEASE OF THE HEART.

V.—TRICUSPID STENOSIS.

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[With Plate III.]

The term tricuspid stenosis embraces all conditions of encroachment on the normal calibre of the tricuspid orifice, whether the obstruction is of a degree to afford any clinical manifestations or no. In this paper, however, I propose to consider only those higher degrees of stenosis, which must, in the nature of things, produce symptoms and signs of their own, elusive though they may be. It is commonly believed and commonly stated that tricuspid stenosis is a very rare, perhaps the rarest of all valvular lesions. There is no justification for this belief, if we accept the wide definition given above, though high degrees of obstruction, comparable with those frequently found at the mitral orifice, are admittedly very rare. As I have shown in a previous article, a trivial degree of tricuspid stenosis is found in nearly a quarter of all cases of mitral stenosis. On the other hand, tricuspid stenosis, without associated affection of other orifices of the heart, is a lesion of the very rarest occurrence. Nine years ago, Herrick collated from various sources 154 cases of tricuspid stenosis, and his analysis shows that only in 12 cases was the morbid process confined exclusively to the tricuspid orifice. The relative frequency of its association with lesions of other orifices is worthy of note:—

Tricuspid stenosis alone - -	12 cases.
Tricuspid and mitral stenosis - -	96 cases.
Tricuspid and pulmonary stenosis -	3 cases.
Tricuspid, aortic, and mitral stenosis -	39 cases.
Tricuspid, aortic, mitral, and pulmonary stenosis - - - -	1 case.
Tricuspid, mitral, and pulmonary stenosis - - - - -	2 cases.
Tricuspid and aortic stenosis - -	1 case.
Total - - - -	154 cases.

Pitt, in 87 cases of tricuspid stenosis collected from the records of Guy's Hospital, found mitral stenosis also in all but 2, and in 1 of these 2 the mitral valves were thickened.

Besides the rarity of the pure lesion and the frequency of associated mitral stenosis, these figures in bulk bring out clearly the common relation to aortic stenosis. Herrick records it in 41 out of 154 cases, and Pitt in 24 out of 87 cases. Pitt also notes thickening of the aortic valves in another 25, so that there was some affection of the aortic valves in 49 out of 87 cases, considerably more than half. In the 7 cases appended to this article, there was associated mitral stenosis in all but 1, and in that it looked as though the dilatation of the rigid orifice might have been a late effect. In 5 of the 7, there was aortic stenosis as well.

The belief that tricuspid stenosis is usually of congenital origin is dying hard, although such evidence as we have is entirely opposed to this view. In the very large proportion of cases, there is a post-natal history of one of the common causes of acute endocarditis. Rheumatic fever is the chief causal agent. Herrick found an unquestionable record of rheumatic fever in no less than 51 out of 154 cases, or, to put it more strikingly, in 51 out of the 57 cases in which a definite cause was noted. Bedford Fenwick's series of 46 cases gave a history of rheumatic fever in 50 per cent.

In my 7 cases, 5 had definitely had rheumatic fever, 1 had had rheumatism, and the remaining case, with no record of rheumatic fever, showed at the autopsy signs of recent acute endocarditis.

Other observers add to the list of causes chorea, typhoid fever, scarlet fever, puerperal fever, and overstrain. As a general rule, then, we may say that there is nothing in the causation of tricuspid stenosis materially different from that of mitral stenosis.

A congenital origin affords no explanation of the markedly greater incidence on the female sex, for statistics of congenital heart disease show that there is no predilection for either sex. Herrick and Lendet's combined statistics of tricuspid stenosis give 114 women to 32 men. Bedford Fenwick's 46 cases included 41 women and 5 men, while of my 7 cases, 6 are women.

The age at which death occurs, in the large majority of cases, namely the third and fourth decades of life, is quite inconsistent with a congenital origin. Contrariwise, in cases in which the congenital origin is assured, death is seldom delayed beyond 4 or 5 years, and far more often occurs in the first few months of life.

Again, reasoning from the analogy of congenital stenosis of the pulmonary origin, and the physical conditions by which, in this condition, the circulation works out its own salvation, we may unhesitatingly assert that if tricuspid stenosis were of fœtal origin, we should habitually find patency of the foramen ovale as a channel of relief. This is not the case; whereas, in cases of tricuspid stenosis of undoubted congenital origin, patency of the foramen ovale is the most frequent of the associated anomalies of arrest.

The frequency with which tricuspid stenosis is combined with mitral stenosis, has led others to maintain that it is secondary to mitral stenosis, and a consequence of protracted high pressure on the mitral valve: the fact that the mitral constriction is almost invariably the more advanced lesion has given some colour to this belief. The arguments against this view, too, are strong. It must have fallen to the lot of most pathologists, in examining the hearts of patients dead after a severe recent acute endocarditis, to see on the tricuspid valves a fringe of small soft vascular vegetations, indicating that the inflammation has attacked simultaneously both sides of the heart. It is not improbable that this is a frequent occurrence, but that whereas on the left side there is a tendency to persistence, on the right side there is a tendency to resolution. To this general rule, however, there are many exceptions, as is indicated by the frequency with which mitral stenosis is associated with some low degree of tricuspid stenosis. Can it be that the clue to the persistence and progress of the mitral lesion, and the disappearance or arrest of the tricuspid lesion is to be found in part in the more yielding musculature of the right auriculo-ventricular orifice?

If back pressure from the left heart were the chief cause of tricuspid stenosis, we should expect to find at least some such association, though in a less degree, with mitral regurgitation, in which the strain on the tricuspid valve may be as great as in mitral stenosis, but such is not the case. Moreover, tricuspid stenosis is at times found at so early a stage of mitral stenosis, as to preclude the probability of its being due to protracted strain, and, per contra, to strengthen the contention that both lesions should be referred to a common cause. In the light, however, of what we know of strain as a cause of valvular disease, it would be foolish to deny some influence to the mitral lesion, and it is at least reasonable to assign a secondary influence in hindering the natural tendency of the right heart to resolution to the exchange of a relatively low pressure for a relatively high pressure, consequent on mitral stenosis.

There is no doubt, also, that induration and shrinking of the tricuspid valve do exceptionally arise as evidence of a widespread degenerative process, more particularly in association with renal cirrhosis and emphysema, and sometimes without affection of the mitral valve : the constriction is then much less than in cases of inflammatory origin.

We conclude then, that tricuspid stenosis, like mitral stenosis, is, in the large majority of cases, an acquired lesion of inflammatory origin, and that, though vascular strain has some influence on the permanence of the tricuspid lesion, the cases, in which strain is the immediate cause, are few and far between.

The post-mortem findings in tricuspid stenosis, seeing that nearly all the cases arise out of acute endocarditis, vary for the most part with the duration of the disease. Pitt found adherent pericardium in 38 out of 87 cases, and recent pericarditis in 10. The 7 cases of this paper showed adherent pericardium in 3, recent pericarditis in 1, and in the remaining 3 the pericardium was normal. This is an even greater frequency than I have shown to be the case with mitral stenosis alone, suggesting, perhaps, that simultaneous affection of both auriculo-ventricular orifices is referable to greater intensity of the initial infective process.

I have already mentioned the fringe of soft gelatinous vascular vegetations that may, at times, be seen on the auricular surface of the tricuspid valve in cases of recent inflammation. In the severely infective cases, these are replaced by bunches of soft exuberant vegetations on the surface of the valve, often spreading to the mural endocardium. The vegetations of recent acute endocarditis, even when large, are so soft and yielding as to produce, as a rule, no serious stenosis, but only incompetency.

In cases of longer standing, sclerotic changes will have followed on the initial exudative process: the valves will be found to be hardened, at first in patches, later throughout their whole extent. Contraction accompanies hardening, and the chordæ tendineæ and musculi papillares are involved in the changes. The degree of induration and contraction is much less than is the case with mitral stenosis, and, only in exceptional cases, is the orifice reduced to the dimensions of even a moderate degree of mitral stenosis. The cases, in which the orifice does not admit two fingers, are very few and far between. With the larger auriculo-ventricular orifice on the right side of the heart, one would expect this to be the case. The high degrees of obstruction of this orifice are almost necessarily the consequence of adhesion of the contiguous borders of the cusps: such is by no means necessarily the case at the smaller left auriculo-ventricular orifice, and herein may be another partial explanation of the infrequency of the higher degrees of tricuspid stenosis. In those that I have seen, the valve forms a membranous diaphragm with central aperture, commonly more or less oval in form or irregularly oblong; a small circular foramen is occasional, but I have not seen an instance of its depression into the ventricle at the apex of a cone, as may be seen at times with mitral stenosis: nor is the crescentic slit, so common in mitral stenosis, found on the right side of the heart. Calcification is far less marked in advanced cases than in mitral stenosis, and at most the valve is flecked with calcareous patches.

In the appearance of the congenital form of tricuspid stenosis, there is little to distinguish it from the acquired form: indeed, when it has arisen out of fœtal endocarditis, the morbid changes are identical, arising as they do from one and the same cause: the orifice tends to become a simple fibrous ring of varying calibre, nearly always associated with some other developmental defects, such as patent foramen ovale, perforation of the ventricular septum, patency of the ductus arteriosus, deficient size of one or other chamber of the heart, and stenosis of other orifices.

One need only mention in passing the one or two records of cases, in which tricuspid stenosis has been due to a cardiac polypus. The changes in the heart consecutive to tricuspid stenosis are in agreement with recognised laws. The right auricle and auricular appendix are always dilated, and, at times, there is evidence of hypertrophy as well. Hypertrophy, however, does not attain to even the degree seen in the left auricle in mitral stenosis, on account of its lesser muscularity. On opening the chest, one may see a tense rounded purple swelling, as large as a tennis ball, occupying the place of the right auricle. The highest degrees of dilatation are found where the constriction is greatest, and co-existent hypertrophy is, at any rate, more apparent when the constriction is less. The coronary sinus has at times been found dilated as well. The endocardium of the right auricle shows, in a less degree, the same greyish-white thickening of the mural endocardium as is apt to arise in the left auricle in mitral stenosis.

In considering the changes in the right heart, one must not forget the relative frequency of adherent pericardium, nor, in the case of the right ventricle, the associated mitral stenosis.

The condition of the right ventricle is usually one of mixed hypertrophy and dilatation, as is the case with the left ventricle in mitral stenosis. In face of the associated mitral stenosis, one would not expect to find the small or normal ventricle that sometimes lies in front of a narrow mitral orifice. In records of cases of uncombined tricuspid stenosis, I have found a few instances of a right ventricle of normal size, but more in which it is described as enlarged, possibly because of increased aspiratory activity.

An old tricuspid lesion may become the site of an acute malignant endocarditis, but there is no exceptional liability to this.

In two cases of tricuspid stenosis, not included in the records appended to this paper, I have met with clotting in the left innominate, internal jugular, and subclavian veins.

The condition of the lungs in these 7 cases, in all of which there was some affection of the mitral orifice, was that of passive congestion of greater or less degree: the frequency of pulmonary apoplexy and hydrothorax is also noteworthy. Pleural adhesion, too, is common, as is the rule when the pericardium is adherent. If mitral stenosis were absent, it would be reasonable to look for the same anæmia of the lungs as in pulmonary stenosis, with the same liability to the invasion of tuberculosis. I can find, in the recorded cases of simple tricuspid stenosis, only one instance of pulmonary tuberculosis.

The condition of the abdominal and general venous circulation is one of stagnation, with the same congestion of the abdominal viscera as in advanced mitral stenosis, and with a greater tendency to subcutaneous dropsy.

The most constant symptom, as in all cases of valvular disease, is dyspnœa on exertion, increasing gradually until it is persistent even with the patient at rest: orthopnœa is common, and, as the disease drags on to its close, paroxysmal attacks of urgent dyspnœa, accompanied by cyanosis, and at times severe præcordial pain: 3 of the 7 patients actually died in such a paroxysm, and, in 2, Cheyne-Stokes respiration ushered in the end.

Cyanosis in some degree and persistent in character is frequent: it chiefly affects the lips, ears, face and extremities, but, in severe cases, the whole body surface. Clubbing of the fingers was present in 1 of the 7 cases, and this appears to represent about the frequency of its presence in recorded cases. Chilliness was not noted at all, but this is frequently a cause of complaint, as is often the case in congenital heart disease. Purpura has also been recorded in some instances, and has been considered to possess diagnostic significance. In a condition, in which the functions of the lungs are so seriously hampered by congestion, cough will tend to be a constant annoyance, while the frequency of infarction will find expression in attacks of pain in the chest with bronchitis and blood-stained expectoration. As the right auricle breaks down, the familiar signs of backward pressure in the systemic venous system set in: the prominence of subcutaneous dropsy, prior to the terminal stage of the illness, may at times suggest the presence of tricuspid stenosis, when all other features are masked by the associated mitral stenosis. The ankles and feet are swollen, and dropsy gradually mounts upwards: the urine is scanty, and high-coloured, and albuminous: the liver is enlarged, and the conjunctivæ acquire a subicteric tint: ascites is the rule. Mackenzie of Burnley has recorded pulsation of the liver in 5 verified cases of tricuspid stenosis, and has demonstrated its auricular character. To him, also, we owe most valuable and precise observations of the actual condition of the jugular veins in tricuspid stenosis. In dilatation of the right heart with incompetence of the tricuspid and venous valves, there is typically a double jugular pulsation, auricular and ventricular. In tricuspid stenosis, he found that the ventricular wave is cut off by the constriction of the orifice. So long as the auricle can contract vigorously, there is a wave produced by, and synchronous with, auricular systole; but, with extreme dilatation of the right auricle, its walls are almost paralysed, and the auricular wave may become so faint as to be imperceptible. I have seen the jugular veins, in undoubted cases of tricuspid stenosis, turgid and so motionless as to suggest the possibility of thrombosis having occurred, but one would fancy that an exceptionally high degree of stenosis must be necessary to cut off the ventricular wave.

The radial pulse presents no constant or characteristic features.

In those rare instances, in which the heart itself exhibits signs indicative of tricuspid stenosis, one reasonably expects to find a close parallelism to the signs of tricuspid stenosis; but, when one regards the great disparity of driving power of the right and left auricle, and the fact that, even on the left side of the heart, the physical signs are apt to diminish or disappear with increasing weakness, it is not surprising to find that the signs of tricuspid stenosis are, as a rule, conspicuous only by their absence. Even if present, it will be readily appreciated how completely such faint evidence would be masked by the signs of the co-existent mitral stenosis, which are commonly so pronounced and prominent.

The area of cardiac dulness is increased in each direction, but the narrower the tricuspid orifice, the larger will be the right auricle, and the wider the dulness to right of the sternum. In the presence of mitral stenosis, the upper limit will also be raised, and there will be epigastric pulsation transmitted from the large and powerful right ventricle.

In a few instances of tricuspid stenosis, verified by post-mortem examination, a presystolic thrill has been detected close to the sternum. In one patient, a harsh vibratory presystolic thrill was felt at the apex, and a second faint purring thrill alongside the sternum. Among my tracings of presystolic thrills, in cases of mitral stenosis, I have some extending to the right of the sternum; one is tempted to believe that such extension may indicate co-existent tricuspid stenosis. In some of these cases, the tricuspid valve was hardened; but there was neither the degree of narrowing of the orifice, nor the hypertrophy of the auricle, that one would regard as essential to the production of a thrill. It must be conceded, as a general rule, that tricuspid stenosis does not give rise to a palpable thrill.

As with the thrill so with the murmur, though commonly not to be detected, there is no question that it has been identified in life in cases of verified tricuspid stenosis. Its site is in the tricuspid area over or close to the sternum. Bearing in mind the relative strength of the right and left auricles, one would expect such a murmur to be soft and low, and such is the case, though records are to be found of comparatively loud murmurs.

From what has been said, it will be apparent how difficult is the diagnosis of tricuspid stenosis. In the 117 cases collected by Lendet, the diagnosis was made only 6 times in life; in the 7 cases recorded below, its existence was not suspected. On the other hand, most physicians will have met cases, in which they have felt assured of the existence of tricuspid stenosis, but have not had the opportunity of verification. It is of little practical value to decide on what evidence one is justified in making a diagnosis of tricuspid stenosis, inasmuch as the lesion is almost never uncombined. The question to answer is rather how, in the presence of mitral stenosis, one may obtain reasonable evidence of the co-existence of tricuspid stenosis. Either lesion will, as a rule, be a consequence of rheumatic fever, and the patient will nearly always be a woman, not older than the fourth decade of life. With mitral stenosis, there will be the quick thrust of the apex beat, a presystolyic thrill and murmur at the apex, and accentuation of the pulmonary second sound, with or without reduplication. To these signs, tricuspid stenosis may add a feebie thrill and murmur in the tricuspid area over or close to the sternum. Notable enlargement of the right auricle, and, in advanced cases of tricuspid stenosis, this should be demonstrable by trans-illumination of the chest, would supply a further link. The combination of mitral stenosis with aortic stenosis should also suggest the likelihood of tricuspid stenosis. The back-pressure signs of mitral stenosis show a close resemblance to those of tricuspid stenosis, but subcutaneous dropsy prior to the stage of final breakdown of the heart is suggestive of tricuspid stenosis. A turgid motionless condition of the jugular veins, and still more in the later stages, a single auricular pulse, either in the jugular veins, or in the liver, will be a strong point in the diagnosis. Cyanosis is certainly more marked and more constant than in simple mitral stenosis. and dyspnœa more urgent and apt to be paroxysmal. In congenital cases of tricuspid stenosis, the variability of the associated lesions makes it impossible to define any general principles of differential diagnosis.

Prognosis, as in all cases of valvular disease, is a matter of the greatest difficulty. The outlook in congenital cases is very grave; some infants live only a few weeks, and scarcely any exceed 4 or 5 years. In the acquired disease, death is commonly before 40; the average at death in my series is 35 years, Duroziez gives 42 years for the low and 32 years for the high degrees of constriction. In any individual case, prognosis will depend on the severity of the back pressure, and the power of response to methods of relief.

With tricuspid stenosis, as with mitral stenosis, treatment is concerned far more with the relief of resistance than with increasing the power of the heart. The heart will need rest: it may even need soothing, if excited; but, if the strain of back pressure is removed, the heart will be in a fair way for a time to work out unaided its own salvation.

Abstract of Cases of Tricuspid Stenosis, from the Records of King's College and the Royal Free Hospitals.

1. E. T., female, 32; 10 days in hospital.

Rheumatic fever at 15. Total abstainer. Had been short of breath on exertion since rheumatic fever. Also liable to cough, and lately to blood-stained expectoration. All the troubles increased by 5 successive pregnancies, so that, for 5 years, she had been unfit for work, but only took to her bed 2 days before admission to hospital. Amenorrhœa for 4 months, and swelling of feet and legs for 3 weeks.

Condition on Admission.—Cannot lie down in bed. Cyanosed. General anasarca. No clubbing of fingers. Heart's apex beat not visible, but felt in the 5th space 4 inches from mid-sternum. Pulsation visible over front of heart and in the epigastrium. No venous pulse detected in jugulars. Area of cardiac dulness reaches 3rd costal cartilage above, 1½–2 inches right of sternum, and 1 inch external to nipple. Presystolic thrill at apex. Presystolic and systolic murmurs at apex. Pulmonary second sound accentuated. Pulse regular and low-tensioned. Artery not thickened. Œdema of bases of both lungs. Liver one finger's breadth below costal margin. Some ascites. Urine, albumen ·7 per cent.

Progress. During stay in hospital, patient suffered from attacks of paroxysmal dyspnœa with cyanosis and severe precordial pain. The

PLATE III.

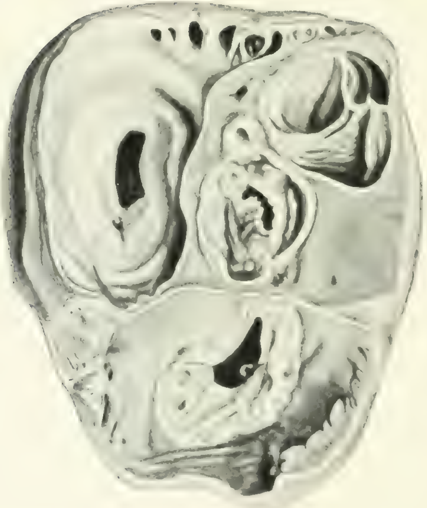

Fig. 1. *Heart of Case 2, showing tricuspid, mitral and aortic stenosis (reduced to $\frac{3}{4}$ of actual size).*

legs were incised to relieve the dropsy, but without much effect. Patient was actively delirious for 24 hours, and sank rapidly and died.

Post-mortem.—Right border of heart, 1½ inches to right of sternum. Apex in anterior axillary line. Heart appears to lie more transversely than normal. Pleuro-pericarditis where the left lung overlaps the heart, due to pulmonary infarcts. Heart, 16 oz. Right auricle much dilated and distended with clot, which extends into the right auricular appendix, where there is also some ante-mortem clot. Tricuspid orifice hardly admits two fingers. The edges of the valves are adherent and shrunken. Patches of calcareous change. Pulmonary orifice and valves normal. Left auricle much dilated and hypertrophied. Mitral orifice a tight button-hole admitting not even the tip of the little finger. Aortic valves, normal. Left ventricle, small. Each pleural cavity half full of fluid. Abundant infarcts in each lung. Kidneys, no obvious change.

Condition.—Tricuspid and mitral stenosis.

2. C. B., woman, 46; in hospital 3 days.

Complains of dropsy. Rheumatic fever at 16. Moderate in liquor. For some years subject to winter cough, but now much worse than before. Six weeks ago began to feel ill and her legs started swelling. This gradually spread to her abdomen so as to keep her in bed. Has suffered much from pain in the abdomen and legs, and over the heart, and great shortness of breath. Complete loss of appetite and persistent headache.

Condition on Admission.—Pale puffy face. Drowsy. Slight œdema of both feet. Lungs, emphysematous. Diffuse bronchitic rales. Heart, vigorous impulse in 5th intercostal space, ½ inch outside nipple. Dulness reaches to upper border of 3rd costal cartilage, the right border of the sternum, and anterior axillary fold. Systolic murmur all over cardiac area and conducted up to the vessels of neck. Pulse irregular, of low tension. Abdomen full of fluid. Urine, thick and scanty; only 5 oz. in first 12 hours; much albumen; granular casts; abundant urates. Sp. gr. 1028.

Progress.—While in hospital was subject to attacks of urgent dyspnœa and cyanosis. Acutely delirious before death, and died in a paroxysm of dyspnœa.

Post-mortem.—4 oz. clear fluid in subdural space. Brain œdematous. Pericardium universal firm adhesions. Tricuspid orifice admits one finger, cusps united, much sclerosed, forming a rigid ring. Right auricle much dilated and hypertrophied, also right ventricle. Mitral orifice button-hole, admits tip of one finger. Edges of valves united forming a rigid and thickened ring almost cartilaginous in hardness. Changes at aortic orifice still more marked. Valves so calcareous and distorted as to admit only point of scissors. The thickening actually encroached on the pulmonary orifice, of which the valves are normal. Left ventricle enormously hypertrophied, some dilatation. Left auricle much dilated, not much hypertrophy. Vessels free from atheroma. Lungs firmly adherent on both sides over large area. Bases œdematous, rest indurated and pigmented. 2 oz. clear fluid in each pleural cavity. Abdomen, 10 pints of clear fluid; great omentum and peritoneum slaty-grey in colour; some fibrous adhesions round spleen. Spleen much enlarged, tough and dark, containing numerous infarcts recent and old, some very large. Weight 15 oz. Liver dark, tough, advanced nutmeg, 61 oz. Alimentary tract shows evidence of chronic venous congestion. Kidneys; several recent infarcts in right kidney, old organised scar in left; passive congestion marked; no diminution of cortex. Right kidney, 6 oz. Left kidney, 4 oz.

Condition.—Tricuspid, mitral, aortic stenosis.

3. M. L., female, 46; 19 days in hospital.

Complains of swelling all over and shortness of breath. Has had rheumatism, but not rheumatic fever. Two months ago swelling of feet and abdomen set in, and has steadily increased. The face and hands are swollen in the morning. For last few days breathlessness has been very severe.

Condition on Admission.—Patient very breathless. General anasarca. Heart's apex beat in 6th space 3 inches outside nipple line. Cardiac dulness reaches 3rd costal cartilage above, right border not defined. Visible pulsation at apex and in epigastrium. At apex systolic murmur conducted into axilla. At base systolic and diastolic murmurs. Lungs, impaired resonance over whole right lung; dulness at both bases. Liver felt one hand's breadth below costal margin. On left side of abdomen, a round and very movable swelling slips beneath the fingers. Urine smoky, much albumen, hyaline and granular casts.

Progress.—Patient suffered from recurrent attacks of urgent dyspnœa. At the end patient became actively delirious, and died suddenly in an attack of dyspnœa.

Post-mortem. Both pleura much fluid. Lungs collapsed. Pericardium, no excess of fluid. Heart, 16 oz. Great hypertrophy, but slight dilatation of both ventricles. Tricuspid valve, shrunken and rigid; orifice stenosed. Mitral valve, thickened and shrunken; orifice stenosed; aortic orifice stenosed. Valves greatly thickened and very rigid. Aorta shows signs of early atheroma. Liver smooth; capsule thickened and puckered in places; nutmeg congestion; much fluid in abdomen. Kidneys, multiple cysts; some of great size, surface pale and rough, but capsule stripped easily; cysts do not communicate with pelvis.

Condition.—Tricuspid, mitral, aortic stenosis.

4. E. A., female, 20; in hospital 17 days.

No history of rheumatism. Complains of breathlessness on exertion. Cough and swelling of legs. For five years shortness of breath and amenorrhœa. For four months, distressing cough and swelling of legs.

Condition on Admission.—Patient cannot lie down. Pulse, 110, irregular and of low tension. Heart's apex beat in 5th space. 1½ inches external to nipple; presystolic and systolic murmurs at apex. Lungs filled with scattered rales. Liver just below costal margin. Urine, $\frac{1}{7}$th albumen. Three days after admission double aortic murmur heard.

Progress.—The main features were obstinate vomiting, and severe attacks of breathlessness. Towards the end the attacks of breathlessness were accompanied by severe pain in the left side radiating down the left arm. Later, the breathing took on Cheyne-Stokes rhythm. Drowsiness preceded death, which occurred in one of the paroxysms of dyspnœa and pain.

Post-mortem. Right auricle and ventricle occupy whole anterior surface of heart, a mere strip of left ventricle being visible. Right auricular appendix passes to left of mid line. Left auricular appendix completely hidden. Tricuspid orifice admits two fingers; mitral orifice only tips of little finger. Two small polypoid masses near fossa ovalis. Aortic valves atheromatous and show two prominent masses of vegetation from recent endocarditis. Heart, 14 oz. Both lungs signs of long standing congestion. Infarct in lower lobe of left lung. Left kidney, two recent infarcts. Right kidney, one old infarct. Liver, 51 oz.

Condition.—Tricuspid, mitral, aortic stenosis.

5. E. L., male, 22; in hospital 4 months.

Complains of pains in the chest and the joints. Two years previously rheumatic fever and pericarditis. Two months ago severe pain in the back of the chest compelled him to leave work, but gradually got better and resumed work. Three days ago severe pain set in in the hips, knees, and ankles, with shortness of breath, cough, and swelling of the left foot.

Condition on Admission.—Apex beat forcible, in 6th space, 1 inch external to nipple. Slight diffused systolic thrill at apex. Dulness reaches the 3rd costal cartilage above and 1 inch to right of sternum. Loud systolic murmur at apex conducted round to the back. Second sound not heard at apex. Pulmonary second sound accentuated. In aortic area systolic murmur conducted up to vessels of neck; also diastolic heard best in 3rd left space and conducted downwards. Pulse, 100. Lungs normal. Liver, 1 inch above umbilicus.

Progress.—Patient was subject to constant attacks of pain in the chest with palpitation, and increasing shortness of breath, with cyanosis. The lungs became œdematous, and there was general anasarca. For a few days before death, he was delirious and finally fell out of bed dead.

Post-mortem.—Face and limbs swollen. Each pleural cavity much fluid. Diffuse pleural adhesions on each side. Pericardium, universal firm adhesion. Heart, 32 oz. Right auricle dilated and hypertrophied. Right ventricle much dilated, some hypertrophy. Pulmonary orifice normal. Tricuspid orifice narrowed; on one cusp a raised semitransparent nodule; another cusp firmly adherent to the ventricular wall. Chordæ tendineæ fibrous; one bundle near the adherent cusp very short and attached to the ventricular wall by a thick fibrous ending of the columna carnea. Left auricle enormously dilated, much hypertrophy. Left ventricle greatly dilated and hypertrophied. Aortic valves thickened. No stenosis. Mitral orifice much thickened. Orifice dilated. Valvular changes very chronic. Abdomen, one pint of blood-stained fluid. Liver, nutmeg. Kidneys, large, pale, firm. Spleen, large, firm.

Condition.—Tricuspid stenosis.

6. A. H., female, 32; 5 days in hospital.

Complains of swelling of the legs and pain over the heart. Rheumatic fever in childhood. Has suffered from swelling of the feet and legs on and off for years. Three months ago a return of pain and swelling of the joints, soon followed by great swelling of the legs and shortness of breath. For eight weeks urine very scanty and red in colour. Lately headache, sickness, and dimness of vision.

Condition on Admission.—Face puffy. Œdema of legs and back. Clubbing of fingers. Urine, 1020; much blood and albumen. Epithelial casts, blood and granular forms. Lungs, œdema of both bases. Heart, apex beat 5th space internal to nipple; dulness one finger's breadth to right of sternum and 3rd costal cartilage above; doubtful systolic murmur at apex. Pulmonary second sound accentuated.

Progress. Three days after admission, sudden incontinence, soon followed by twitchings of the face and unconsciousness; left arm and leg became paralysed; some spasmodic movements on right side. Patient did not recover consciousness. Cheyne-Stokes respiration set in, and death ensued rapidly.

Post-mortem.—Pericardium normal. Heart, aortic valves adherent and thickened; stenosis of orifice. Mitral orifice admits the tip of one finger; also shows signs of recent endocarditis. Tricuspid ring rigid, admits tip of two fingers. Lungs, œdematous; adhesions of right pleura. Liver, smooth, large, fatty; 71 ozs. Spleen, many adhesions; one recent and two old infarcts; 2½ ozs. Kidneys, slightly granular; enlarged and congested. Brain, in middle two-thirds of right hemisphere area of hæmorrhage, triangular on section; convolutions much flattened; appeared to be a hæmorrhagic infarct. A few hæmorrhagic spots in cortex anterior to the chief hæmorrhage.

Condition.—Tricuspid, mitral, aortic stenosis.

7. S. P., female, 48; 50 days in hospital.

Complains of shortness of breath. Acute rheumatism at 18. Moderate in liquor. Symptoms of heart failing date from eight years ago, since which time frequently in hospital. For last four months increasing shortness of breath and swelling of feet.

Condition on Admission.—Pulse, 84; slightly irregular. Artery not thickened. Some præcordial prominence. Apex beat in 6th space 1 inch external to nipple; dulness to 3rd costal cartilage above and 1 inch to right of sternum. Presystolic thrill at apex; presystolic and systolic murmurs at apex. Epigrastric pulsation. Œdema of bases of both lungs, and of legs. Liver down to umbilicus: ascites. Urine, 1020; much albumen.

Progress.—Seemed to improve at first, but subsequently anasarca spread and reached the abdominal wall. Was subject to attacks of dyspnœa with cyanosis; also severe pain in abdomen. Finally developed hydrothorax and died.

Post-mortem.—Œdema of legs. Double hydrothorax; pleural adhesions over both lungs, which are congested and emphysematous. Signs of old pericarditis and some blood stained fluid in the pericardium. Right auricle much dilated; right ventricle as thick as left ventricle and much dilated. Mitral orifice admits one finger. Chordæ tendineæ and musculi papillares thickened. Tricuspid orifice admits three finger tips. Middle and right aortic valves small, contracted, and adherent to each other; remaining valve normal Liver showed red atrophy, a good deal contracted, having a rather cirrhotic appearance on section. Lobules stand up clearly, veins dilated. Kidneys, red granular appearance. Cortex small, capsule adherent. Spleen, small and fibrous.

Condition.—Tricuspid, mitral, aortic stenosis.

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POSTPARTUM HÆMORRHAGE.

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It is probable that the main impression left upon the mind of any reader by the perusal of Dr. Fitzgerald's paper in the November number of THE PRACTITIONER, so far as it purports to be an answer to my own in the August part, will be that, if the advocates of the old methods of treatment of postpartum hæmorrhage have no better arguments to bring forward in their defence, they are indeed in a very sorry case.

For what do the arguments therein contained amount to? There is—

1st. A suggestion that these methods and the reasons for them are not understood, or properly carried out by their opponents.

2nd. The assertion that these methods have proved their efficiency in competent hands.

3rd. A misstatement of what has been said in the former paper.

4th. A repetition of the author's belief in a force which he calls "Retraction."

5th. Doubt is expressed as to the influence of position upon the flow of blood in the veins.

6th and last. A claim that the method advised in the August paper can only be carried out in hospital.

Something might perhaps be said of the questionable taste with which these "arguments" are set forth, but that is for Dr. Fitzgerald's consideration. I do not propose to imitate it, and shall simply reply to these points, for it is of great importance that those, to whom the lives of many women will be entrusted in the future, should not believe that a few words of almost contemptuous reference disposes of the question as to how they may best meet the most momentous crisis of their professional lives, when they are confronted with it.

It must be remembered that postpartum hæmorrhage is essentially a general practitioner's tragedy. I have never seen a case in hospital, and one or two other consultants with much larger experience than mine of hospitals for women, have assured me that they, too, have never seen such cases in hospital practice. Indeed, they can, by the nature of things, be extremely rarely seen, if at all, by those whose experience has been limited to those institutions. That, however, does not prove that they do not occur. Every general practitioner of large experience knows them, and understands only too well the methods advised for their treatment, and the reasons which are supposed to justify such measures. It is upon the fact that I was myself for nearly twenty years engaged in such practice that I base my own claim to speak, and it is out of the experience so gained, that I offer to my confrères the advice given in the August number. Dr. Fitzgerald evidently considers the cases generally to be as rare as he has found them in the Rotunda. Playfair, on the contrary, quotes the Report of the Registrar-General for the five years from 1872 to 1876, in which 3,524 deaths are attributed to flooding. "The majority of these must have been caused by postpartum hæmorrhage" (Playfair). My friend, Dr. Le Page, finds in the same reports for 1905, under the same heading, over 800 deaths reported; practically the same number. This means that, in the British Islands alone, over 2 deaths per day occur from these causes. Surely this is sufficient to satisfy even Dr. Fitzgerald as to its frequency; but, if such cases only happened once a month, or once a year, that would be sufficient to justify an earnest attempt to put an end to what is, in the opinion of many of us, an entirely preventable loss of life, due mainly to erroneous teaching upon this subject.

Probably this fact is the best possible confirmation of the latter part of Dr. Fitzgerald's paper, in which the dangers of improper conduct of the third stage of labour are emphasised. In a hospital, this stage will be well managed, and, therefore, cases of this kind are not so likely to arise in such a building. That they will never get there, when once they have happened outside, is the experience of all those who have had the misfortune to encounter them.

The practitioner in charge of such a case will do all in his power to check the unexpected, disconcerting, and, to his mind, disgraceful loss of blood, before calling in any other help. I use the word "disgraceful," because, though at first sight it looks misplaced, it most exactly describes the feeling of the accoucheur. For several years, he has been able to boast that he has never lost a case; now, for the first time, he sees a woman, who has trusted him in consequence of that reputation, slipping through his hands; and doing so from sheer loss of blood which he is powerless to check. She was apparently a healthy woman when he entered the house; she had merely to go through a purely physiological process; when he leaves, she is dead. Can it be wondered that he feels it a disgrace? And the consequences in his practice soon prove that others also regard it in the same light.

It need scarcely be said that there are many factors and considerations at work in general practice, which have no force at all within the walls of a hospital, and of which a man, who has never been in such practice, can have but the most elementary idea.

As I said, the medical attendant will do everything he has learnt before he will confess that he is powerless; and events march quickly at such a time. It is not a case of days, scarcely of hours. By the time during which he has been over the round of remedies which are given in every text-book, the woman is well nigh pulseless. Let anyone who wants a graphic account, which yet is admirably true to nature, of what happens, read Playfair's description.[1] When this condition is reached, and it does not take long, it is impossible to think of removing the patient. She would die before the end of the street was reached. No, the attendant must either rely upon his own resources, or he must call in the aid of the nearest man who happens to be at home; thus consultants rarely see these cases even in their private work.

And when the nearest practitioner arrives, what more can he do than has already been done by his colleague? He, like his professional brother, has been taught the same things; the same list of possible but never certain remedies; he may repeat one of these experiments, may possibly remember one which the other had forgotten, but he cannot feel certain of success, for the men who taught him, the authors of text-books on which he relies, did not feel certain themselves; both these men are left, at this acute moment, with nothing more definite than the advice that "if he finds that he is not succeeding in checking the bleeding with one manœuvre, he may pass to the next at once, holding the uterus bimanually whilst the materials necessary for the next are being prepared" (Dakin). "Supposing these means fail and the uterus obstinately refuses to contract in spite of all our efforts, and, do what we may, cases of this kind will occur" (Playfair). "In the worst cases the methods may fail. Plugging the uterine cavity may be done—after bimanual compression has been applied without complete success—if it," the plugging, "fails to stimulate the uterus, it probably does little good, for, owing to the distensibility of the elastic uterine walls, it is practically impossible to pack the organ so tightly as to control hæmorrhage by direct pressure" (Eden). The writer of the article in the November number appears to be scandalised by my assertion that those, who teach these methods, do not believe in them. What else can be said of men who advise, at such a time, that a series of experiments should be made, the result of which they do not guarantee, when both the reputation of the accoucheur and the life of the patient depend upon them?

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[1] Playfair: *Science and Practice of Midwifery*, Vol. 2, p. 121.

After such statements as these, taken from recognised text-books, what becomes of his second assertion that these measures have proved their efficiency in competent hands? Does he then not consider that Playfair, Dakin, and Eden, to mention no others, are competent men?

"To teach students the causes of postpartum hæmorrhage, he says, is but to increase their uncertainty and anxiety, when brought face to face with this complication. Teach them no causes he suggests, for they are too many." The "he" refers to myself. This is Dr. Fitzgerald's reading of my appeal to teachers of midwifery in the August number to teach their students, to burn into their memories the one and only cause that is of practical use to them at such a time: that the uterus bleeds because it is tired out, and, therefore, *cannot* contract; that it imperatively demands rest; that, if it can obtain that rest, it will inevitably contract again and to good purpose; that if, in consequence of too great dependence of the medical attendant upon the mere dicta of anyone, he persists in flogging the uterus in a vain attempt to make it contract in its present exhausted state, he may obtain some flickering useless attempt at contraction, which will only have the effect of still further exhausting the uterine fibres, but he will not succeed in stopping the hæmorrhage, and that this will in every case be the natural result, because of the *cause*, exhaustion, which is only temporary, of the uterine muscle; that, if he can tide over the time required by that muscle for rest, his patient will live; that in consequence of that being the *cause*, the hæmostasis must be brought about by other means than by contraction of these exhausted fibres, which it is for the time, impossible to procure; that the time for an academic discussion of the many ways, in which this "exhaustion" may have been brought about, is not then when his patient is bleeding to death, but later, when, the danger over, he sits quietly at home, and has ample leisure to define and redefine to his heart's content; but that, whilst his patient's life hangs on his action, he should concentrate his whole attention on the one only effective cause: exhaustion.

I think that all unprejudiced readers will agree that Dr. Fitzgerald's version of what I there said can scarcely be described as accurate.

It is almost with a feeling of relief that one turns to the next point, the discussion of a force, which Dr. Fitzgerald calls "Retraction." One is inclined to congratulate him on its discovery, but it might be well if he would christen his new force by some other name, so as to prevent inevitable confusion; there already exists something known as "Retraction," which is mentioned and described by most other writers on this subject, but which has evidently nothing to do with his discovery. Fothergill says (*Manual of Midwifery*, p. 184), "After each contraction there is relaxation, but this is not complete, and each muscle fibre, after the temporary shortening and thickening of contraction is over, retains a certain degree of shortening and thickening called 'retraction.' Thus contraction is temporary and transient, whilst 'retraction' is permanent and progressive." Dakin says (*Handbook of Midwifery*, p. 97), "Retraction enables a (muscular) fibre, which has shortened, to relax without returning to its original length." Galabin defines "retraction" as "the contraction and shortening of the uterine muscle, not followed by relaxation."

Dr. Fitzgerald says that his force has nothing to do with "contraction." It is distinct from and entirely different to contraction, and therefore is "entirely different to and distinct from" the "retraction" referred to by the authorities I have quoted: "It is," he says, "the force which moulds the uterus upon its contents." Several rather curious results appear inevitably to be involved in this definition; Dr. Fitzgerald's "retraction" evidently must be mainly observed whilst the uterus has any contents; it should, therefore, be at least present, if not most completely evident during the whole of pregnancy; it probably will disappear when it is most wanted, that is when the uterus has just been emptied of its contents, or at the end of the third stage. This is unfortunate, because it is just then that it is needed; however, if it is gone there is no use regretting it; we must contrive to get along without it. This force is, however so novel and interesting that I trust Dr. Fitzgerald may be induced to tell us something still more definite about it.

The next point is not one which admits of much discussion. The influence of position upon the blood in the veins is so well known, that at the present time to "doubt" it is much the same as suggesting that astronomers may be wrong in believing in the rotation of the earth.

But inasmuch as Dr. Fitzgerald's last contention may influence some who have not tried the method, to believe that, as he says, compression of the aorta and elevation of the pelvis can only be carried out in hospital, it may be well to point out that my experience of this method is entirely gained from its practice in private houses, and that not a single case was treated in hospital; that the properties (to use an actor's term) required, are simply a bed, a table, and the bare fist of the operator; and that these are obtainable without recourse to anything that cannot be found in the smallest cottage in England.

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THE INTERPRETATION OF SPHYGMOGRAPH TRACINGS, AND OF TRACINGS PRODUCED BY COMPRESSING THE BRACHIAL ARTERY.

THE FACTORS WHICH ARE INVOLVED IN THE PRODUCTION OF ANACROTISM.

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Fellow of University College, London.

I.—INTRODUCTORY.

During a six months' residence at University College Hospital, my attention was directed to the clinical instruments in general use for the estimation of the state of the circulatory system. A prolonged series of observations, then and since, has shown the possibilities of the instruments, and has also demonstrated the complexity of the results obtained with one of them, the Dudgeon's sphygmograph. In this communication, the other instrument referred to is Dr. C. J. Martin's modified Riva Rocci Sphygmomanometer.* The Dudgeon's Sphygmograph has been chosen, not only because it is widely used for clinical purposes, but because, from my experience of it, I consider that it is an instrument of considerable delicacy and accuracy, and that it compares favourably with appliances of its kind. Out of a collection of over a thousand tracings, a few are published as illustrative of this paper.

II.—PRINCIPLES OF THE DUDGEON'S SPHYGMOGRAPH.

a. *The Forces acting on the Pad.*—The pad of the Dudgeon's sphygmograph rides between two lateral standards and has a slightly convex surface. When the instrument is applied, it is acted upon by forces represented in the diagram, (fig. 1). It is pressed upon by a steel spring from above (force A), and by certain forces from beneath. The forces pressing on the pad from beneath are somewhat complex. The blood in the radial artery is under a certain pressure which can be estimated with a small error; for the time being, let this force be considered unvarying (represented by the arrow E). This force is usually opposed by an equal and opposite force D, representing the tension of arterial wall and surrounding tissues. With the

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* Supplied by Hawksley, London.

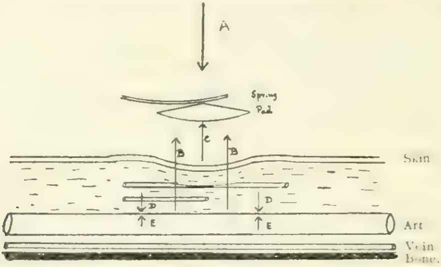

Fig. 1.

application of the sphygmograph the force D is diminished, and the resultant of the forces D and E is now opposed to the pad, and may be represented by the force B. If there is no indentation of the artery by the pad, that portion of the force D dependent upon arterial wall tension is unaffected. It is improbable that the pad ever indents the vessel to any extent in taking tracings of pulse beats, beyond causing slight flattening of it; but even slight flattening of it will considerably decrease D, and magnify B to a corresponding extent. A considerable pad pressure would entirely abolish D, so far as it depended on the arterial wall. Such a pad pressure rarely occurs in the practical application of the instrument, as will be subsequently seen. In the meanwhile a new force C has arisen, owing to the resistance of the tissues to the depression of the pad. This force depends upon the various tissues compressed, first and most important the skin, and with greater pressures, underlying tissues, and eventually the arterial wall itself. The arrangement of the pad is such that, though a certain amount of resistance is met by the centre of the pad, the greater resistances are met by the proximal and distal edges of it, where most surface distortion occurs, (fig. 2).

Were there no friction between pad and skin, tissue resistance would act on the pad in the directions *a*, *a*. Given the extra factor, friction, a certain amount of tangential pull is added (*b*). The resultant of forces *a*, *a*, *b*, *b* may be represented by *C* acting through the centre of the pad. The force *b* acts parallel to the line of the vessel; laterally it is abolished by the pressure of the standards.

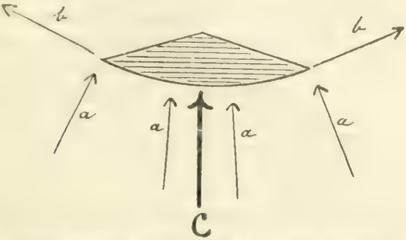

Fig. 2.—*Diagram illustrating the action of tissue resistance on the pad. Section running parallel to the artery.*

We are now in a position to consider the factors influencing the forces involved. The subject is one of considerable importance and interest, for a consideration of it enables the rational interpretation of various tracings from the same pulse. With uniform pressures on the pad, the spring obeys Hooke's Law, (tracing 1). As the pressure of the pad on the skin (force A) increases, B and C will each increase. At any instant, when the levers are at rest, $A = B + C$. The force C, however, will increase out of proportion to the increase in A. It is the prevailing opinion that animal tissues do not obey Hooke's law, though this view has recently been criticised by Haycraft,[13] and Nicholls[29] concludes that an artery obeys it within the natural limits of distension. It matters not whether Hooke's Law is obeyed or otherwise by a particular tissue, for, as the pad sinks, it will constantly meet with new resistances. It is for this reason that the force C increases out of proportion to A. Consequently the force B will not increase in the same proportion as A. The tissue resistance C will vary with each application of the instrument, and the rate of increase will also vary. The point of importance remains, that, with increased pad pressures, the resultant pad depressions become smaller in proportion, owing to the rapid and disproportionate increase of opposing tissue resistance. It will be impossible, with the spring pressures practicable, to place any great pressure on the wall of the artery.* When large spring pressures are employed, so much tissue depression occurs, and the changes of tissue resistance from point to point are so great, that the pulse excursion becomes very much diminished. The changes in the tissue resistances are more rapid during diastolic phases of a pulse beat, for during these phases there is more pad depression. It consequently follows that, in all pulse tracings, the systolic phases of the curve are exaggerated in excursion, (in the ordinate line), and the diastolic phases are decreased.† In this respect those pulse tracings are chiefly affected which are obtained from individuals with a large interval between systolic and diastolic pressures. In the normal pulse the effect is slight, in high tension pulses almost negligible, for here the force B increases very rapidly. In low tension pulses and aortic regurgitant pulses, there will always be a great tendency towards curtailing of the diastolic phases of the curves.‡ The flattening of the diastolic portions of a curve in aortic regurgitation, with increased pad pressures, has been referred to briefly by Galabin,[12] Roy and Adami,[31] Mahomed,[26] and Marey.[27] It is essential in such cases that several tracings should be taken, or that that tracing be chosen as most typical which shows the diastolic phases of the beats to most advantage, (tracing 7). By this procedure, a truer idea of the pressure changes is obtained. It is also essential that the lightest possible pad pressure should be used. If, on the other hand, the pressure is so light on the skin at diastole that the greater part of it falls on skin only, the artery may fail to affect the pad at this stage, and diastolic phases of the beat will not show, (*see* tracing 5). A good excursion is therefore also requisite. It does not follow that the maximum excursion will give the truest record, though this may generally hold, (tracing 7). Frequently a curve at lighter pad pressure and of slightly less excursion magnifies the diastolic phases in proportion to the systolic. It is clear also that every effort should be made to reduce tissue resistance. The artery for investigation must be as superficial as possible. The radial at the wrist is perhaps the most suitable. The advantage of underlying bone lies in the fact that the depression of the skin by the standards is least when it is present. For this reason it is probable that truer records are often obtainable from the superficial temporal artery. Tissue resistance at the wrist can be decreased by extending the joint, so that the surface of the skin becomes convex, and the tangential pull on the proximal and distal edges of the pad is diminished.

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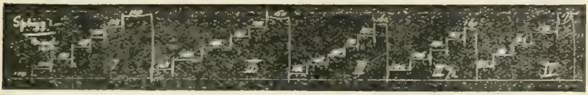
* The pressure placed on the artery necessarily varies with the conditions of the experiment; when a tracing is properly and carefully taken it never approaches diastolic pressure at diastole, unless this be exceptionally low.

† The proportions of the ordinates are influenced by other factors, and to a varying extent in different pulse types; reference has been made to this in communications to the *Journ. of Physiol.*

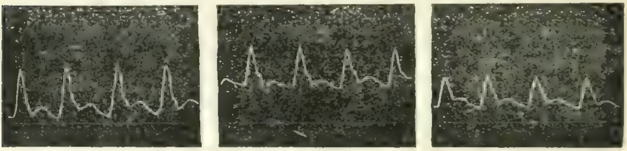
‡ There is always the possibility with extremely low diastolic pressures that the artery will collapse, and the pad meet the resistance of bone.

A great many efforts have been made to accurately measure the pressures on the pad in mm. Hg., necessary to raise the writing point to certain definite lines on the paper. For this purpose, an artificial schema has been employed, connected with a Hg. manometer and a small balloon resting under the pad. The attempts have been abandoned, as it has not yet been found possible to obtain an apparatus free from fallacy and giving uniform results. The nearest approach to accuracy was obtained with a small balloon of rubber, coated with peritoneal membrane, and with this it could be shown that the spring and levers approximately obey Hooke's law. A tracing obtained with this instrument is given, (tracing 1); the figures indicate pressures in mm. of Hg.; the Roman numerals spring pressures; they are only accurate in the comparative sense. It is probable that the pressure of the pad on the artery, (force B), can be estimated approximately by producing venous congestion in the arm to definite pressures (*see*[23]). This is accomplished by distending a Riva Rocci armlet on the upper arm to a definite pressure, and allowing the venous blood to collect to the full. The force B may be considered a fairly uniform one, as the space over which it acts is, to a large extent, enclosed by the standards, pad, and artery, and there is a thickness of skin and tissues

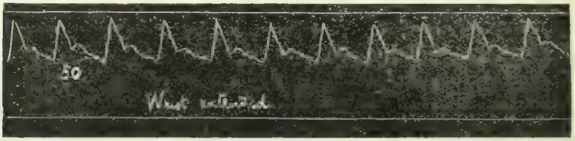


Tracing 1.

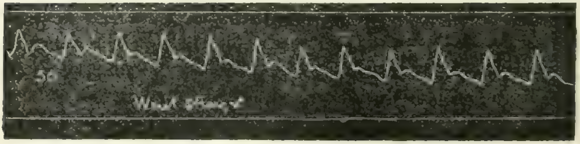
intervening, which tends to the distribution of pressures. When the sphygmograph is applied to the wrist, underlying veins will be collapsed, and will re-open and affect the height of the tracing, only when the venous pressure has risen to the height of the pressure of the pad on the artery. It is essential, in these estimations, that the instrument should be fixed to the wrist by weight extension, as venous congestion affects the tracing plethysmographically when a band fixation is used. By this method, it has been calculated that the pressure necessary to obtain a maximal tracing, from a patient with normal blood pressures, is usually between 30 and 50 mm. Hg., and that a pressure on the artery of 100 mm. Hg. is very rarely, if ever, employed.*



Tracing 2, *a.* (40.)† *b.* (60.) *c.* (80.)



Tracing 3.



Tracing 4.

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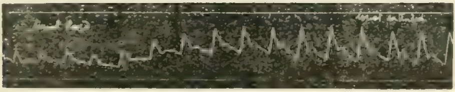
* It is generally believed, *cf.* [16] [19] [27] and [28], that maximal pulsation is given at diastolic pressure. This is only so if circular pressure is adopted, for a full account of which *see* Janeway.[20]

† The number in brackets indicates the pressure of pad on artery in mm. Hg.

For purposes of illustration, tracings are given from subjects with normal and low arterial pressures, and also from cases of aortic regurgitations. Tracings 2 (*a*, *b* and *c*) are from a normal pulse (systolic blood pressure 130, diastolic 90, as estimated by a method similar to that of Erlanger[9]). The tracings show the gradual obliteration of diastolic phases of the beats. The differences in a normal pulse are necessarily slight. The venous congestion, necessary to affect the systolic portions of these tracings, was 40, 60, and 80 mm. Hg. respectively. These are approximately the pressures exerted by the pad on the artery at systole.

Tracings 3 and 4 illustrate the diminution of tissue resistance consequent on the extension of the wrist joint. The first portion of the tracing, (tracing 3), shows the effect of the release of a venous distension of 50 mm. Hg. with the wrist extended. It shows that the pressure on the vessel at diastole was equivalent to more than 50 mm. Hg., for the tracing is unaffected. The second portion of the tracing, (tracing 4), shows the same experiment with the wrist straight. The effect is marked on both systolic and diastolic phases of the tracing, which falls away rapidly. The pressure of the pad on the vessel, in this instance, was therefore a good deal less than 50 mm. Hg. at systole. Tracing 5 shows the marked increase of diastolic phases in the radial pulse, on passively bending the wrist from the flexed to the extended position. It illustrates the same principles. The pressure on the artery is too light with the wrist flexed for the diastolic portions to be recorded.* Tracings 6 *a*, *b*, *c*, were taken from a dicrotic pulse at spring pressures marked I, II, and IV. respectively, on the instrument employed. The effect on the dicrotic wave is very striking. The effect of increased pad pressure on high tension pulses is very small. Roy and



Tracing 5.

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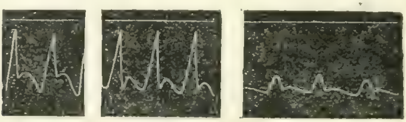
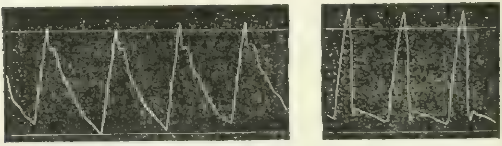
* The reverse of this is sometimes found where the diastolic pressure is low and pressure greater.

Adami[31] and others give tracings illustrating it. The best curves are obtained with large pressures (*cp.*[33]). Lower pressures may result in the obliteration of diastolic phases. It has been said that high tension pulses may yield anacrotism with increased pressures.[3 4] This is not the experience of these investigations. Pulse waves of high tension are, as a rule, the most difficult to convert into anacrotic waves. It is the pulse curve, with a sustained outflow wave, which is easy to convert, as will be seen later. The effect of increased pressure on an aortic pulse is more definite. Here the most favourable conditions are present for the curtailing of the diastolic phases of the beat. Tracings 7 and 8 (*a* to *f*) are good examples of this effect, (*cp.* tracings 26 and 31). The same is shown in tracing 9, in which the tension of the band encircling the wrist was gradually reduced. The effect of venous congestion on the aortic pulse is somewhat irregular. As a rule, both systolic and diastolic phases are affected as the pressure rises* (tracing 10). Often the diastolic portion



Tracing 6, *a*. *b*. *c*.



a. (20.) *b*. (60.)

Tracing 7. (*The pressures marked were obtained by the method described.*)

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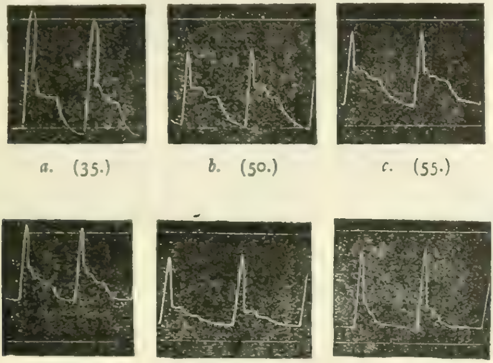
* The amount of venous congestion is given on the tracing in mm. Hg. (*i.e.*, 0, 10, 18, 20, 0).

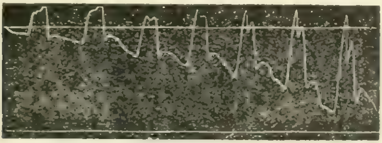
of the tracing shows more displacement than the systolic, (tracings 11 and 12, in which the venous pressures are given).



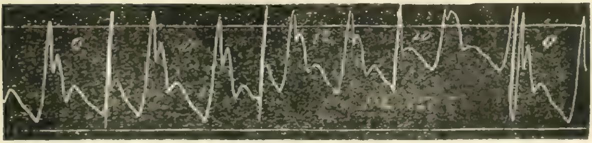
a. (35.) *b.* (50.) *c.* (55.)

d. (60.) *e.* (80.) *f.* (85.)

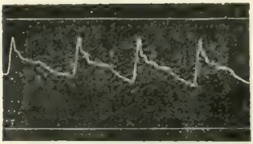
Tracing 8. The pressures given were calculated by the method described, and were at diastole. B.P. systolic 130, *diastolic* 60.



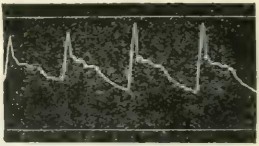
Tracing 9.



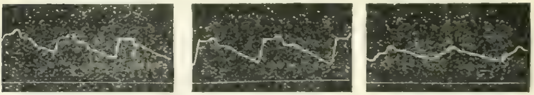
Tracing 10.



Tracing 11. (*Venous pressure reduced from* 55–0.)



Tracing 12. (*Venous pressure reduced from* 50–0.)



Tracing 13. *a*, (15). *b*, (35). *c*, (60).

Finally, a tracing is given (No. 13) showing the effect of pad pressure on an anacrotic pulse. It will be seen that the tendency to anacrotism is enhanced. This tracing will be referred to subsequently.

The tracings, which have been given, illustrate the fact that pulse tracings, when considered as curves of pressure, must be interpreted with very great care, and that conclusions, drawn from the relative positions of ordinates of the curves, may be entirely misleading. Conclusions drawn from the abscissæ are also not without fallacy (*see* tracings 7 and 13).

III.—THE EFFECT ON A PULSE TRACING OF OCCLUSION OF THE BRACHIAL ARTERY.

Method.—The only references* to this method of investigation, which have been found, are a short paragraph in an article by Hill, Barnard, and Sequeira,[17] and an article by Hill,[15] which is an extract of the former work. The conclusion drawn is, that by this means we have a method of investigating the peripheral circulation; a rapid and steep fall in the tracing indicating a high arterial tension, a low venous pressure, and a rapid escape of venous blood. A note is added by Hill,[15] that, in making such observations, the maximum pulse excursion should be obtained, and the sphygmograph should be on a level with the heart. In all the tracings accompanying this communication, the brachial artery has been the seat of compression, for, in the middle of its course, it is usually easily occluded, movements of the arm do not accompany the compression, if care is used, and the tracings are not confused by the inflow of blood from collateral anastomosis.* The arm is gripped between fingers and thumb, the latter resting lightly on the artery. Compression takes place against the bone, and occlusion is practically instantaneous. The sphygmograph is placed on the wrist, with the pad resting on the radial sulcus, and is arranged to give a tracing in the centre of the smoked paper. It is adjusted at a pad pressure,† which gives, on occlusion, the maximum fall of the lever.‡ This maximum fall is obtained on the end of the recording paper, and when obtained, and giving a uniform drop with successive brachial compressions, a tracing is taken. First a few natural pulse curves are obtained, then a compression curve, and finally a few more pulse curves, acting as a control, and showing any dislocation of the tracing produced by movements of the arm, if they should occur.

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* A single tracing is given by François-Franck; Marey's *Physiologie Expérimentale*, Tome II., p. 26, fig. 7.

Theoretical Considerations.—Briefly, tracings taken in this way are an index of the rate at which the artery collapses under the pressure of the sphygmograph pad, for the instrument records changes in antero-posterior diameter § of the vessel upon which it is laid. These changes depend on the internal pressures of blood in the vessel, and the external pressure of the spring pad. By external pressure is signified, not the pressure on the skin, but the pressure on the artery, (opposing the force B). The amount of fall during an experiment depends on the diameter of the vessel and the amount of original indentation of it by the pad. The rate of fall depends on the same factors, and in addition to the changes in internal and external pressures. Now the changes in arterial pressure, during the experiment, depend upon the arterial pressure, venous pressure, state of the peripheral vessels, and the viscosity of the blood. A high arterial pressure, a low venous pressure, dilated arterioles, and decreased viscosity, will tend to greater velocity of outflow, and a steeper fall in the tracing. After occlusion of the brachial artery, the arterial pressure falls, consequently the curve traced will tend to be concave upwards. It has been shown that the external pressures do not obey Hook s Law, and consequently the curve given is not an exact index of the rate of fall of pressures in the artery. Were it possible to attach a light lever to the arterial wall itself, the amount and rate of fall could be accurately estimated without difficulty, as the complicating factor of tissue resistance would be avoided. Hill, Barnard, and Sequeira[17] considered that the main structures to be taken into account are the venæ comites. From the experiments made on these veins,[23] it has been concluded that they only influence the pad when the blood in them is at unnaturally high pressures, (if the brachial artery is not interfered with). When the pressures in the artery are reduced, venous distension will influence the tracing to a greater extent. The increasing tissue resistances, as the pad sinks, will tend to give a curve concave upwards, as does also the falling arterial pressure. It will also be obvious that, when the compression takes place at the end of a pulse beat (tracings 15–17, etc.), a continuation of the natural fall of the pulse curve may be expected. The chief value of this method of investigation lies in the prolongation of the diastolic phases of the curve, and the influence of various factors upon these phases is the more easily inquired into. It will now be more clearly understood why the diastolic phases of a pulse curve are reduced in size, and that the natural fall of a pulse beat curve is materially influenced by tissue resistances. It is only possible to make a comparison, and, for this reason, it is essential that a standard should be fixed. Reasons have been given why, in choosing typical tracings, the height and excursion of diastolic phases of the beat above a base line should be taken as of first importance. The same arguments apply to the choosing of a compression tracing. It should in each case be maximal, and it will tend to be maximal when the diastolic phases of the pulse curve are maximal. It also follows that factors, which influence the diastolic portions of a pulse curve, will similarly affect the compression curve. The amount of a compression fall may be estimated from the apices or bases of the beats. In future, these lines will be referred to as the "systolic line" and "diastolic line." * A third line drawn through the lowest point of the compression curve will be spoken of as the "compression base line," (tracing 14). This last line indicates that point in the tracing, when the sphygmograph pad is no longer influenced by arterial pressures, but when it is supported by tissue resistances only. When circumstances are favourable, this will indicate a point when the pad meets the resistance of the posterior wall of the artery and the underlying bone. Although it must be admitted that the pulse wave is chiefly a matter of pressures and not of volumes, yet with the application of the sphygmograph the artery becomes subject to further changes, if not of volume, certainly of antero-posterior diameter, and upon this we rely for the graphic record, for the pad follows the anterior wall. This is of importance, for, with high pressure pulses, it becomes necessary to place considerable pressure on the vessel. It is insisted upon by many writers that this pressure should be equivalent to diastolic pressure, an end which they suppose to be attained by recording the maximum excursion. Reasons have been given for doubting this; moreover, there comes the practical difficulty that a maximum excursion cannot always be taken by the paper, and, in cases of aortic regurgitation, could only be overcome at the expense of the essential compactness of a clinical instrument. In the case of such pulses, it consequently becomes necessary to take a smaller, if somewhat modified, tracing, by reducing or increasing the pressures. When the pad

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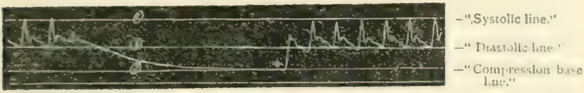
* The interference of aberrant vessels would necessarily influence the results. They are rare, and their presence would be recognised.

† Indicated by Roman figures on the circular disc attached.

‡ In those cases in which, on compression, the tracing falls off the paper, a second tracing is taken at a higher level on the paper.

§ I cannot enter here on a discussion of the nature of a pulse wave as felt by the finger. Broadbent [4] believes that there is little change in the volume of the vessel, but rather a change in shape. Mayo Collier [6] considered that there is an actual enlargement of the sectional area of the vessel. Whichever view is held, a change in antero-posterior diameter must be admitted.



Tracing 14.

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* In preference to the term "respiratory line" employed by Mahomed[46] and Graham Steell.[23]

pressure on the artery rises to or above diastolic pressure at diastole, the artery will collapse at the end of the beat; the greater the pressure, the more complete will be the cutting off of diastolic phases of the curves, and the more sudden will the descent of the writing point be to the "compression base line." (It has been argued that it is possible for the diastolic phases to be curtailed before diastolic pressure is reached, for the pressures exerted on the pad may be so small in comparison with the tissue pressure, that it has little effect on the pad.) It so happens that, when diastolic pressure is low, a compression of the brachial artery has little effect on the height of the pad, and the "diastolic" and "compression base" lines traced coincide. Given the lightest pressure at which this occurs, the pressure on the vessel at diastole is diastolic pressure or a little less, and a new method of estimating this is now possible. In my opinion the method is not sufficiently accurate to give readings in mm Hg. of exactness; but it is of value in identifying pulses, which have abnormally low diastolic pressures. One thing alone is essential; that the pressures, employed in the application of the instrument used, should be approximately known.* As a matter of experience, it may be said that a sufficient knowledge of this is quickly acquired by taking tracings from pulses known to have a low diastolic pressure.

Tracings obtained and the Interpretation of them.—When occlusion of the brachial artery takes place in a normal circulatory system, the falling pressure is indicated in the tracing by a curve concave upwards. The rate of fall can be estimated by noting the time taken for the complete fall to occur, or noting the obliquity of the fall. It is practically impossible to exert such pressure † on the artery of an individual with a normal pulse, that no fall in a tracing occurs on compression. This is natural, for the diastolic pressure approaches the systolic so closely that diastolic phases of the beat cannot be obliterated. If the compression is complete the line traced is even and unnotched. When the vessel is released, the pulse curves following release frequently show a tendency to ascend.

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* The variation in different instruments is very great. The Roman figures indicating oz., troy, are of little value.

† The fact is impressed that pressure on the artery is not equivalent to pressure on the skin.

This is more conspicuous in cases of moderate high tension (tracings 15, 16). It is due perhaps to an increasing tone in the arterial wall,* rather than to an increase in the volume of blood.† Should the release happen at the beginning of a beat, this beat is frequently exaggerated, (tracings 18*a* and 19*b*). Also there is often increased dicrotism of the first beat, *cp.* [17]. (tracings 18*a* and *b* and 19*b*). Should the release happen between the beats, the lever rises to a line corresponding to the pressure of that phase of the pulse wave which takes place at this instant, (tracings 25, 26, 28, and 33). If the excursion is great, there may be some overriding (tracing 20 and 23*b*); the overriding might be attributed to instrumental error, but it must not be forgotten that the portion of the artery, lying between compression point and sphygmograph, forms an essential part of the instrument, and that much, if not the whole of the overriding may occur in this. The same explanation holds for the exaggeration of the first beat referred to above, for the released blood is rushing into collapsed arteries, and its momentum is considerable. The rise on release between the beats is due to the elastic tension in the arterial reservoir above the point of compression. Immediately at the release, a slight depression frequently marks the curve (tracings 15, 16, 18, 19, 21, 22, and 23B), perhaps due to a short negative wave set up by the sudden withdrawal of the compressing force. A slight positive curve similarly marks the onset of compression, (same tracings).

The Effect of Increased Pad Pressure.—An increase of external pressures should tend to produce a fall of the lever diminished in amount, but increased in rate. With a heavier pad pressure, the original indentation of the artery will be greater, and the changes in tissue resistance will proceed more rapidly, when compression occurs. The fall, obtained with the heavier pressure, will correspond to the last part of the fall with the lighter pressure, for the first part of the fall, with the heavy pressure, will have occurred when the spring pressure was altered. In tracings at different pressures, the amount of fall will be decreased in proportion as the pressure is heavier, (tracings 15 and 16). The rate of fall will not appear to be more rapid, if the first portions of these tracings are compared. As has been pointed out, they do not correspond. The first portion of the curve in tracing 16, corresponds with the last part of the curve in tracing 15. The effect on most pulses of increased pressure is to flatten the general down slope of the pulse curve, and this is often the case with compression tracings at high pad pressures. To take an extreme case, if pad pressure on the artery is increased to diastolic pressure, there is no further fall on compression, and the compression line is flat.

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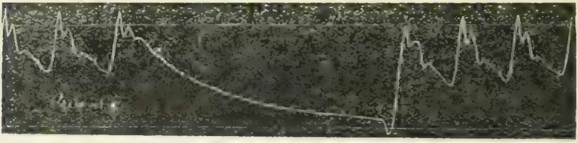
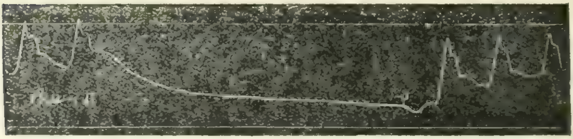
* For a full account of this development of tension in an arterial wall in response to increase of pressure consult Bayliss.[1]

† In view of the curves of François-Franck (Marey's *Physiologie Expérimentale*, Tome II., p. 25, Fig. 6), it is possible that this rise is due to an increased volume rather than to an increase of tension. His observations were first noticed as this paper was completed.



Tracing 15. *B.P.* 160. *Pressure II.*



Tracing 16. *B.P.* 160. *Pressure IV.*

The Effect of Venous Congestion.—This affects the fall in two ways. By increasing tissue resistance, and by increasing the obstruction in the capillaries. It is not possible to separate the two factors. Tracings 17, *a*, *b*, and *c*, demonstrate the main point at issue. In 17, *b*, and *c*, venous pressure was raised to 25 and 50 mm. Hg. respectively. The fall is gradually decreased. Artificial venous congestion frequently cuts off diastolic portions of pulse beats, particularly in aortic cases, as has been shown. The possibility of venous distension affecting the natural pulse must be remembered.* It is this venous interference which prevents the accurate

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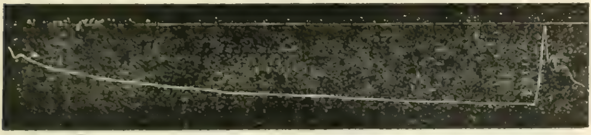
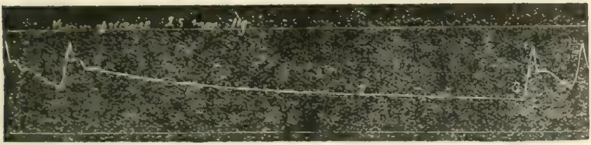
*** As first pointed out by Hill, Barnard, and Sequeira.[17]**

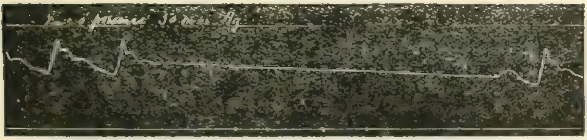
estimation of diastolic pressure by pumping the air in the armlet on the upper arm to a height in mm. of Hg. necessary



Tracing 17 *a.** *B.P.* 140.



Tracing 17 *b.*



Tracing 17 *c.*

to affect the tracing. Unless it is known that the pad of the sphygmograph presses on the artery at diastolic pressure, no idea of diastolic pressure can be obtained.†

Tracings obtained from High Tension Pulses.—In the high tension pulse, where the skin is cool or dry, the drop is moderate in amount and in rate. In cases of cold skin and thin wiry pulse the fall is small, and in spite of the raised pressure is slow. In tracings 18, *a—d*, are given a few examples of a number of compression results taken from a woman admitted to hospital with uræmic dyspnœa. The blood pressures, corresponding to these four tracings, were 250 mm., 230 mm., 200 mm., and 180 mm. respectively. The fall in pressure was due to a venesection of 20 ozs., and occurred gradually within two hours of the bleeding. An increased velocity of the blood is conclusively shown by the gradually increasing obliquity of the fall.* In tracing 18*a*, there is no appreciable fall, though the emptying of the vessel is shown by the decrotic character of the first beat following the release. At this time the patient showed some venous engorgement. When tracing 18*b* was taken there was none. A few days later, when the blood pressure had returned to 235

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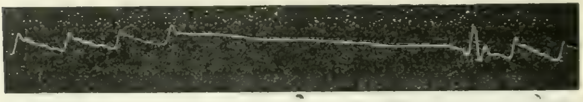
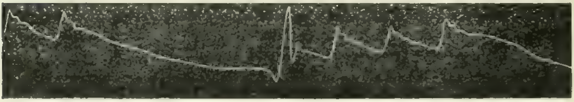
* By an unfortunate oversight the end of this tracing was too much cut. The comparison of the drop in this and the next figure is thereby rendered more difficult, for the measurement of the ordinate between the "compression base line" and the termination of the first new beat is not possible.

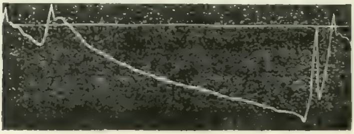
† When the instrument is properly applied, the diastolic phases are cut off much earlier by venous congestion than through direct pressure on the artery by the armlet.



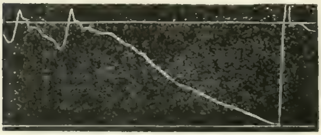
Tracing 18 *a*. *B.P.* 250 *mm.*



Tracing 18 *b*. *B.P.* 230 *mm.*



Tracing 18 *c*. *B.P.* 200 *mm.*



Tracing 18 *d*. *B.P.* 180 *mm.*

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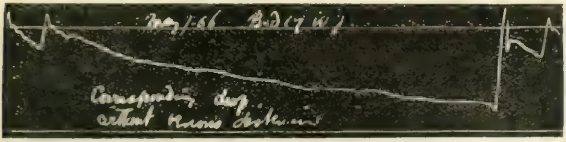
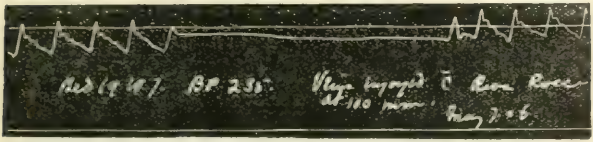
* This increased velocity was probably due to a large extent to alterations in the blood's viscosity, consequent on the bleeding. In experiments, such as these, unless maximal falls are obtained, the comparison is worthless.

mm., a fair drop indicated a persistent increase in velocity, tracing 19*a*. Tracing 19*b* shows the effect of a venous congestion of 100 mm Hg.* on this pulse directly afterwards. It is

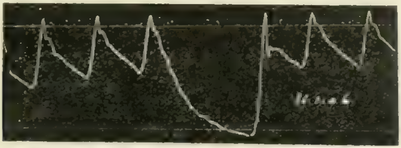


Tracing 19 *a*. *B.P.* 235.



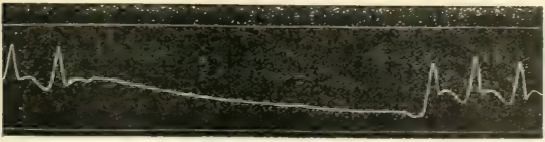
Tracing 19 *b*. *B.P.* 235.

practically a duplicate of tracing 18*a*. The next tracing, (No. 20), is from a case of apoplexy with full bounding pulse and moist hot skin.



Tracing 20. *B.P.* 168.

Tracings obtained from Low-tension Pulses.—As a general rule, dicrotic pulses give small drops, (tracing 21). Dicrotic



Tracing 21. *B.P.* 90. *Boy age* 10.

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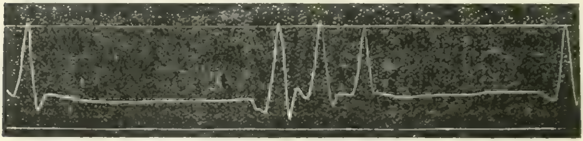
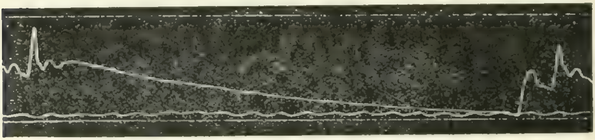
* The pressure used was too great to institute a fair comparison, the effect of venous congestion on tracing 18*a* was probably only very slight.

pulses with a smaller excursion may show larger drops; as a general rule, the higher the blood-pressure the greater the drop. In one instance in which the excursion was great and the blood-pressure low, there was no fall, (tracing *22*).

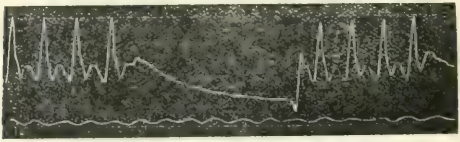


Tracing 22. *B.P.* 70. *Youth age* 21.

The next two tracings are from a normal individual, before and after *2* mins. of amyl nitrite, (tracing *23 a* and *b*). The fall is of equal extent in each case, but takes place in one-third of the time after the amyl nitrite.



Tracing 23 *a*.



Tracing 23 *b*.

Tracings obtained from Cases of Aortic Regurgitation.—In cases of aortic insufficiency, quoted by Janeway,[20] the pressures shown were remarkable. He states that a 75 per cent. rise of the original diastolic pressure with each systole is an average rise, and gives an example of 113 per cent. rise.* In a study of 30 cases of aortic incompetence, in which several hundred compression tracings were taken, a fall below the "diastolic line" was seen in two cases only. In one it was inappreciable. In the other the second sound was present. In the remaining 28 cases the second sound was inaudible. It might be thought that, with very light pressure, a fall would be obtained. If so, it must be a rare event. It is probable that it is prevented, in many cases, by a slight degree of venous congestion set up by the brachial occlusion, a congestion which is occasionally just visible.

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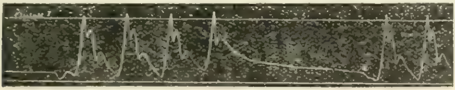
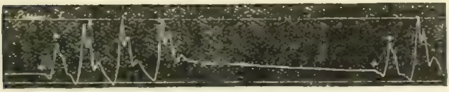
* I have already given a case with a rise of 116 per cent., (tracing 8).

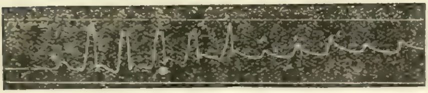
In aortic insufficiency we have the typical "collapsing" pulse; tracings 24 and 25, taken at lightest possible pad pressure, are graphic records of the collapse. The beginning of tracing 26 shows a compression result, from the same pulse, as 25, but at a greater pad pressure; it shows further collapse between the beats. As a rule, in aortic cases, the beats show less tendency to ascent after release. The first beat after release shows generally no exaggeration, and if any only slight. A slight exaggeration has occurred only in those cases, in which the signs indicated less extreme regurgitation, but this fact is mentioned tentatively, for the signs of the quantity of regurgitation are notoriously uncertain, and post-mortems, so far obtained on cases previously sphygmographed, have been too



Tracing 24. *Pressure I.*



Tracing 25. *Pressure I.*



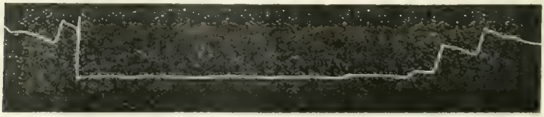
Tracing 26. *Pressure VI.*

limited to justify dogmatic conclusions. The "compression base-line" is frequently higher than the "diastolic line." This is possibly due to some overriding, producing a lowered "diastolic line." It is more probably due to slight leakage through the block at systole, and the cutting off of any centripetal waves,* a question to be more fully discussed later. The relative position of the "diastolic" and "compression base" lines is so striking and constant, in cases of marked aortic insufficiency, that I venture to think it may prove a sign of value in those cases, in which local signs are masked by exocardial sounds, or by overlapping of the lung, and in instances of early diastolic murmurs of mitral origin. I am aware that a more accurate method exists in the determination of diastolic pressure, by instruments now on the market, but they are both too cumbersome and too expensive for the clinician. With this method, all that is necessary is a Dudgeon's sphygmograph.

IV.—THE EFFECT OF PARTIAL OBLITERATION OF THE BRACHIAL ARTERY ON A PULSE TRACING.

Method.—The sphygmograph is first applied, and the upper arm is then lightly gripped as for the complete occlusion. A few natural pulse beats are taken as a control, and the tracing is then modified by increasing the finger pressure on the brachial artery. One, two, or more fingers may be used in the compression.

By this method, an intermitting occlusion is obtained. The systolic wave is modified, and the diastolic portions of the wave are cut off. The mean pressure in the radial vessel is lowered in the normal cardio-vascular system (*cp.* Janeway,[20] p. 190), and with the normal pulse tracing the line of tracing drops. In tracing 27, this fall and the rise on release are shown; the individual beats are seen througho ut



Tracing 27. *B.P.* 120.

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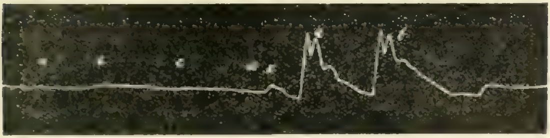
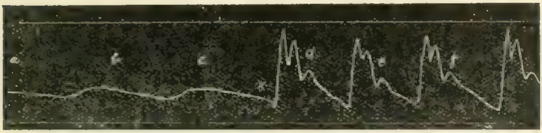
* Or to the production of slight venous congestion.

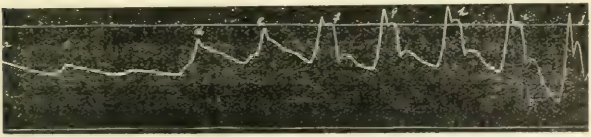
Curves obtained in Cases of Aortic Regurgitation.—The beats produced by partial compression do not fall below the diastolic base line, but ride above it, (tracings 26, 28, 29 and 30); in many cases this happens to such an extent as to indicate a rise in the mean pressure, (tracing 30).



Tracing 28. *B.P.* 160.

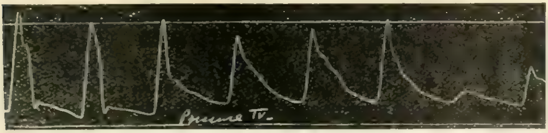


Tracing 29. *B.P.* 156.



Tracing 30.

A further point of some interest is the modification of individual curves. They show marked delay in descent, particularly when sphygmograph pad-pressure is high, and causes some cutting off of diastolic phases in the natural beat.



Tracing 31.

This effect is shown slightly in tracings 26, 29, and 30, and is well marked in tracing 31. It is a manifestation of the elevation of mean pressure already referred to; this elevation is due to the valvular nature of the block, which allows systolic pressures to pass, cuts off diastolic phases, and prevents regurgitant waves. Actual regurgitation is mentioned with reserve, since the only other evidence of its occurrence is the occasional audibility of a to and fro murmur on compressing the vessel with a stethoscope.[8] Reserve is the greater as our knowledge of the production of such bruits is limited (*see* [29]).

V.—THE PRODUCTION OF ANACROTISM.

The literature dealing with anacrotism appears to be scanty, and very much scattered. The conditions, associated with its production, are mentioned by the majority of writers in short paragraphs. So far as can be found, the chief work on the subject is by Mahomed[26] and Graham Steell.[33] Text-book writers vary a good deal in their descriptions of it, and of the factors credited with its production. There appears to be no connected review of the subject. In the following pages, the term "anacrotism" will be used strictly to indicate the tracing of a pulse beat, in which the primary elevation fails to reach the summit of the tracing. The term "anacrotic pulse" will be applied to pulses in which the character of the pulse is recognisable by palpation. The origin of anacrotism may be regarded from two standpoints, the clinical and the physical. Clinically, it is associated by almost all writers, with aortic stenosis, prominent amongst whom are the last two mentioned. Landois and Stirling[22] associate it with stenosis of the aorta, senile arteries, increased peripheral resistance, and hypertrophy and dilatation of the left ventricle. Sansom[32] adds to this list, chronic renal disease, and angina pectoris.

Now the pulse, in aneurysm of the aorta, frequently resembles the anacrotic beat, though the wave is usually flatter. This type of pulse is also found in endarteritis, thrombosis, and embolism,[5] and, according to Sansom, in cases of cardiac enfeeblement, and narrowed systemic capillaries. In aneurysm, the flat-topped pulse may be produced in three separate ways. By an aneurysm, situated on a vessel, between the heart and the artery investigated; by a collection of clot in the origin of the innominate or subclavian vessel, inducing stenosis; or by the pressure of the aneurysmal tumour on either vessel. With the first method of causation we are not here concerned. There can be little doubt that, in the majority of instances, the anacrotic pulse and the flat-topped pulse are similarly produced; any cause tending to the production of the one will, if it is exaggerated, tend to the production of the other, as will be subsequently shown. For practical purposes, the ætiology of both may be considered together. The clinical evidence all points to the production of anacrotism, either through obstruction to the outflow of blood, or to inability of the cardiac muscle to meet the demands made upon it. As high blood pressure produces anacrotism rather as the exception than the rule, it follows that alone peripheral obstruction is insufficient to account for it, and the view held by Roy and Adami[31] is probably the correct one, that, in these circumstances, the additional factor of weakened cardiac impulse aids its production.*

Dealing with the experimental side of the question, we find that physiologists recognise that anacrotism may occur in the curve of intraventricular pressure. Reference need only be made to the very exact curves of Bayliss and Starling.[2] Macdonnell[24] succeeded in rendering a cardiogram anacrotic, in the case of a woman with exceptionally lax abdominal walls, by compressing the aorta. His tracings were taken subdiaphragmatically, and were probably very correct records. Anacrotic cardiograms were also recorded by Roy and Adami,[31] and are indeed of common occurrence. There is a very apparent possibility suggested by these observations, namely, that the arteries, in certain cases, may be so rigid that the anacrotic curve of intraventricular pressure is transmitted as such from the heart.

Passing from the question of general anacrotism, we come to the important subject of its local production. Landois and Stirling[21] mention that it may occur in the stagnating blood of paralysed limbs, and that, after ligature of an artery, the pulse may be anacrotic, when it returns in the distal end of the vessel. It is a matter of common observation, amongst those who have experience of the sphygmograph, that anacrotism may be produced by increasing pad pressure[3 and 4].

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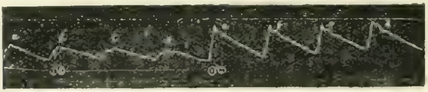
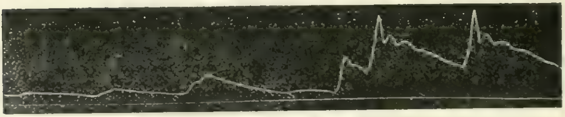
* *Cf.* the observation of v. Frey,[11] who noticed this phenomenon while the heart was escaping from vagal inhibition.

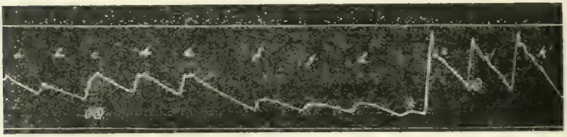
Certain observations with an important bearing on the subject will now be recorded. In taking partial compression tracings, it has been found that many of the "compressed" beats are remarkably anacrotic.* Certain tracings (Nos. 26 and 30), already given, show it. It is most easily produced in pulse curves which already show a prominent "predicrotic" wave (tracings 32, 33 and 34). Its production in this way is parallel to the method of its production, in cases of partial obliteration of an artery by clot, or by the pressure of aneurysms or tumours. Tracing 35*a* is maximal, and was taken on April 19, from the left radial of a patient suffering from thoracic aneurysm; (there was no valvular disease). The right and left pulses on this date were identical. On May 13, the tracing from the right radial showed little change (35*c*), but that from the left was palpably weaker, and gave the curves shown in 35*b*, in which marked tendency to anacrotism



Tracing 32.



Tracing 33.



Tracing 34.

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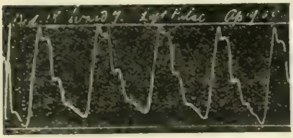
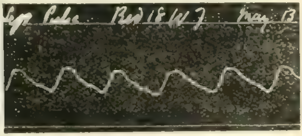
* Since these observations were made it has been found that Landois and Stirling[22] also record this fact.

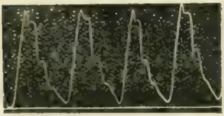
is exhibited. The blood pressures in the two arms were, in the right, 140 mm., in the left, 120 mm., on that date (*cp.* [20]). A partial compression tracing, taken from the right arm at the same time, the last part of which is shown in tracing 35*d*, shows the artificial production of similar curves. In tracing 36***a***, anacrotism is shown in the pulse of another patient with thoracic aneurysm. The left pulse is flat-topped (36*b*). Tracing 37 shows two flat-topped beats produced by partial brachial occlusion in an aortic case. Anacrotism may be produced in normal pulses by this method, and also by



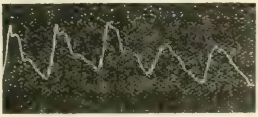
Tracing 35 *a.*



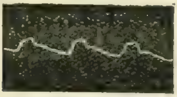
Tracing 35 *b.*



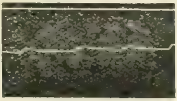
Tracing 35 *c.*



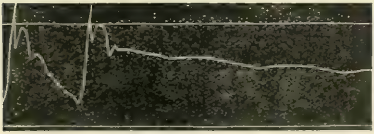
Tracing 35 *d.*



Tracing 36 *a.*

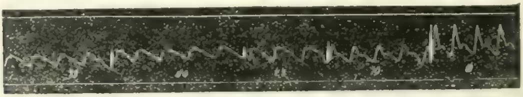


Tracing 36 *b.*



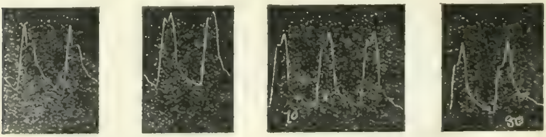
Tracing 37.

wrapping a Riva Rocci armlet around the hand, and raising the pressure in it to, or about diastolic pressure, tracing 38, *cp.* [23].



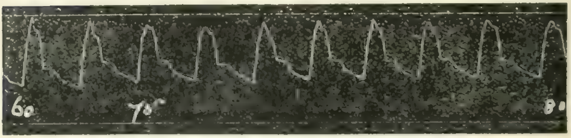
Tracing 38.

In no case has the latter method been attempted on a normal pulse without success. The range of pressures, at which anacrotism may be produced, is small, and in the tracing given is from 75–85 mm. Hg.* The blood pressures of the individual were 115 and 75, syst. and diast. The production of anacrotism in this way is more striking, when the predicrotic is well marked, and when it reaches almost as high as the primary wave. Consequently it is more difficult to produce in cases of aortic regurgitation. Two examples of this are given, one in which the anacrotism is produced from waves already present, (tracing 39), the other in which the primary wave is divided,



0. 50. 70. 80.

Tracing 39.†



Tracing 40.†

(tracing 40). As a rule, heavy pad pressures must be used for its production in these cases. In tracing 43,† is the result of a similar procedure applied to an anacrotic pulse, from a case of aortic stenosis (*cp.* tracing 13). Anacrotism is rendered more distinct. Tracing 42 shows its production by increasing pad pressure on a pulse, in which the predicrotic was practically level with the primary wave. Tracing 41 shows the effect of releasing an armlet from a pressure of 140 to 100 mm. of Hg., on the upper arm, in a normal subject with systolic blood pressure of 130 mm. By this method,

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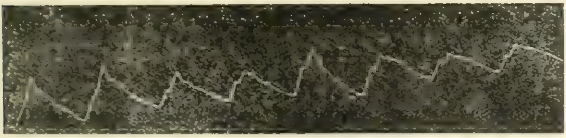
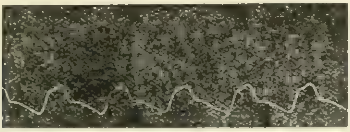
* The pressures, indicated on the tracing, are those in the armlet wrapped about the hand

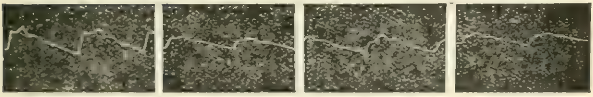
† The pressures, given below the tracings, are those in the armlet wrapped about the hand.



Tracing 41.

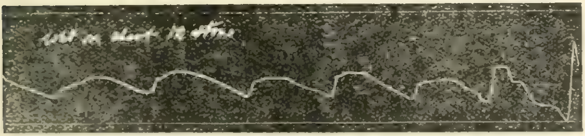


Tracing 42.



0. 50. 70. 140.

Tracing 43.[*]



Tracing 44.

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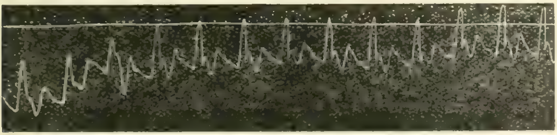
[*] The pressures, given below the tracings, are those in the armlet wrapped about the hand.

venous blood gradually accumulates in the arm as the beats come through, and as the obstruction increases, anacrotism becomes more and more distinct. Finally, an example of general anacrotism is given in tracing 44. It was produced by loading the chest, of an individual with a normal pulse, with a weight of 10 stone.

The literature, dealing with the subject of anacrotism from the physical standpoint, must now be dealt with. In this there is great divergence of opinion. Mackenzie[25] appears to regard the anacrotic peak as an artifact, instrumental in origin. Hill[15] states that it is a wave in which the predicrotic is most prominent. Steell[33] describes it as a beat with lowered "percussion" wave. Broadbent[4] is, I believe, alone in considering it due to the delay of the dicrotic wave from the last beat.* Roy and Adami[31] attribute it to decreased prominence of the papillary wave. These views, with the exception of those of Broadbent and Mackenzie, are merely descriptive of the pulse beat. Roy and Adami, however, discuss the subject more fully. Their "papillary" wave is supposed to have its origin in the contraction of the papillary muscles, and they attribute anacrotism to several forces combined. With a dilated ventricle, the papillary muscles are supposed by them to act to less advantage on the increased ventricular contents. With an increased resistance in front, the ventricle is supposed to be stimulated during its contraction to a more powerful effort (*cp.* [19 and 34]); finally some of the force of the heart's beat is supposed to be lost in overcoming the obstruction. The dependence of the first peak, on contraction of the papillary muscles, has been called in question by the work of Haycraft and Paterson,[14] Fenwick and Overend,[10] and Waller.[35] Landois and Stirling[22] believed that anacrotism, in cases of aortic regurgitation, was due to blood entering from the auricle and passing direct to the aorta. There seems to be little or no evidence for this. Reason has been given why, for the present, the origin of the primary wave should not be regarded as having its origin in contraction of the papillary muscles; but there is every reason to suppose, from the work of Roy and Adami,[31] and others, that it is of intraventricular origin. The origin of the "predicrotic," ("tidal," or "outflow remainder") wave has been

* The usual explanation of the "hyperdicrotic" pulse.

a subject of considerable discussion. The work of v. Kries,[21] who believed it to be a reflected wave, has not been generally accepted. A simple experiment, originally performed by Marey,[27] *cp.* [12] also, practically excludes reflected waves. An obstruction to the outflow of blood from an artery is obtained by occluding the vessel directly below the pad of the sphygmograph. The result of this is to exaggerate each wave on the curve, (tracing 45), and, as a rule, it gives a relatively higher position to the predicrotic, and may even demonstrate a predicrotic, where it was not before apparent. Waves of reflexion from the obstruction will, in these circumstances, return so rapidly to the pad as to produce a summated effect. No waves are ever lost in the tracing.



Tracing 45.

All prominent waves must, for the time being, be regarded as of central origin. Although it is probable that anacrotism is usually produced from pre-existing waves, yet it has been shown (tracing 40), that this is not necessarily the case. In the case of local anacrotism, there is no reason to suppose that there is any alteration in the force of the heart's contraction in the middle of its systole, though the possibility must remain, so far as general anacrotism is concerned. The possibility must also remain that, with an anacrotic curve of intraventricular pressure, the condition of the systemic circuit may be such as to lead to the conduction of this form of curve to the smaller arteries. At the same time, a general explanation for the production of anacrotism, under so many varying conditions, is required. In the first place, it must be recognised that anacrotism may be produced by the damping down of a portion, or the whole of the primary wave, or even the whole of the primary wave with a portion of the second wave. The experiments, which have been quoted and described, all show a common factor. There is an obstruction in the artery, in which the anacrotic wave is produced. It is a matter of indifference as to what the nature of the block is, so long as it is a passable one. A complete block does not lead to anacrotism (tracing 45). At the same time it must be noted that, with obstruction, the "predicrotic" wave frequently tends to hold a higher position in the tracing. This may be explained, in part at least, by the fact that the general fall in the fundamental curve is greater as the waves proceed from heart to periphery, (the raising of the predicrotic is not well shown in the accompanying tracing). It follows that anacrotism will be produced more readily, in vessels near the heart. Again, anacrotism occurs at a point proximal and distal to the block. Its occurrence distally is to be attributed to the fact that a wave alters only slowly, as it is transmitted to the periphery. Its occurrence proximally suggests that the interference with the wave occurs, not at the situation of the block, but at the inlet of the artery which contains the obstruction. Local anacrotism may consequently be attributed to the fact that the pulse wave, as it travels to the periphery, meets with a surmountable obstruction on entering the vessel in which it occurs. Taking the simplest case, that of a distal obstruction, at a pressure at or about diastolic pressure, it will be obvious that the obstruction will only exist at the end of diastole, and the only phase of the pulse wave, which experiences it, will be the primary rise. The primary rise of pressure frees the way for the passage of succeeding pressures. Given a gradually increasing obstruction (tracing 41), more and more of the advancing wave will be affected, and the curve will tend to become more flat-topped (tracing 38).*

I believe this to be the true explanation of anacrotism, that the leading wave pressures, depending on velocities, are partially spent in overcoming obstruction ahead. Applying this argument to the production of general anacrotism in aortic stenosis, a slight dilatation of the ring must be assumed with the commencement of each systole. Should the ring be so sclerosed in any individual as to disprove the possibility of such a distension, the conclusion must be that the intraventricular curve is anacrotic, and that it is conducted as such to the periphery. It has been said that the pressures will be partially spent on the obstruction. If the primary pressures are great, and so excessive as to destroy the normal relationship between them and the resistance to be overcome, as is probable in the case of aortic regurgitation, and in some cases of high tension, then it can be understood that a considerable obstruction will not, of necessity, be associated with anacrotism. An obstruction is, therefore, not the sole essential factor, but also a primary wave of pressure insufficient to cope with it. So it comes about that, while local anacrotism is of little prognostic importance so far as the heart is concerned, general anacrotism may be. In conclusion, the view of Mackenzie,[25] that the first peak is an artifact, is untenable ; it is palpable to the finger, and the delicate measures, adopted by Bayliss and Starling,[2] in recording intraventricular curves, preclude the possibility of its artificial production in this situation.*

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* *Cp.* 23.

CHIEF CONCLUSIONS.

1. Pulse tracings, taken with the Dudgeon's sphygmograph, show disproportion in the ordinate lines. The systolic phases of the waves are exaggerated when compared with the diastolic phases. If several varying tracings are taken from the same pulse, the safest criterion to follow, in choosing a tracing, is the relative excursion of the diastolic phases.

2. Compression tracings are of value inasmuch as they prolong the diastolic phases of a beat, and allow an easier and more certain investigation of the rapidity of the outflow of the blood. They also provide a clinical method of testing the diastolic pressure of a pulse, and are particularly valuable in cases of aortic regurgitation.

3. Several methods of producing anacrotism locally are described. It is probable that the majority of anacrotic pulses, and flat-topped pulses have common factors of origin. The flat-topped pulse is a later stage of the anacrotic. Anacrotism is produced locally, by a loss of the normal relationship between the pressures of the wave entering a vessel, and the resistances encountered in that vessel. General anacrotism depends on the same cause ; in this case there is a special consideration, for the wave may be anacrotic from the start,

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* *See further*, Lewis, *Journ. of Anat. and Phys.*, 1907.

and conditions of the vessel wall, or an obstruction in front, may aid in its conduction as an anacrotic wave.

The researches, on which this article is based, were completed in August 1906.

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INTRA-CRANIAL ABSCESS: WHEN, AND WHEN NOT TO OPERATE.

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We are told that there is a tide in the affairs of men, which, if taken at the flood, leads on to fortune, etc., and the same idea holds good with regard to disease, and very especially is it so in the case of abscess of the brain. The precise time when it is most judicious to operate, when success will be most assured, requires more than a little discrimination.

Before entering upon the subject proper of this paper, I desire to state definitely that I do not propose to discuss the *locale* of the disease, or the relative value of symptoms, subjective or objective, except in so far as they form part of the diagnosis, which deals with the most opportune moment for operation; and it is assumed that the diagnosis of the abscess, condition, and also its *locale* have been thoroughly established.

It is understood that no surgeon, of his own will, will wait an hour after making the diagnosis of intra-cranial abscess, before he proceeds to operate. In addition to other weighty reasons, the possibility of his patient's sudden death must always be vividly before him. He will remember that abscess in the temporo-spheroidal region may burst its possibly thin containing wall, and flood the lateral ventricle with its contents, and thus cause sudden stoppage of the heart and respiration: also that abscess of the cerebellum may cause death by its pressure on the respiratory centre, respiration ceasing some time before the heart. Many conditions, however, may prevent his arriving at a definite diagnosis, and he may perforce allow an operable second stage to pass into a hazardous third; friends may be unwilling to give their consent; the headache may not be of the kind he has observed in previous cases; the vomiting may be very slight, or, in rare cases, may be altogether absent. Again, there may be no evidence of paralysis of cranial or other nerves, or he may be holding back, because there is as yet no optic neuritis. This may prove to be a fatal mistake on his part, for the production of optic neuritis, appreciable by the ophthalmoscope, is a work of time, of weeks rather than days. That incipient inflammation of the optic nerve may be coincident with the earliest inflammatory condition of brain matter is not unlikely; but before the inflammation has extended so far along the nerve as the entrance of the latter into the eyeball, thus proclaiming its presence to the ophthalmoscopist, intra-cranial abscess may be more than ripe for the surgeon's knife; while, therefore, the presence of optic neuritis may support the idea of operation, the absence of it must not deter the surgeon from operating. On the other hand, the possibility of optic atrophy following on after an intense optic neuritis, due to intra-cranial abscess, need not be entertained. The patient may be quite blind, but the period of the continuance of the intense optic neuritis is relatively so short that an operation, which saves life will also in all probability restore the sight in a remarkably short time. The result of operation for tumour is sometimes lamentably otherwise.

Probably no symptom, considered by itself, is more impressive in the study of the stage of abscess formation than the state of the pupils. Whatever may be the headache, the vomiting, the paralytic state, or the state of the reflexes, if the pupils respond quickly, all idea of pressure may be ruled out. When, however, they have become sluggish, or, what is worse, when there is absolute loss of reaction to light and accommodation, the most favourable moment for operation is rapidly passing away. When they are contracted and insensitive, or dilated and unequal, the danger of the patient's sinking, a few hours after operation, is very great. Some cases may possibly do well when operated upon with the pupils in either of these conditions, but the majority do not.

Rigors are not very frequently observed, but when they do occur, in conditions unassociated with sinus thrombosis, they suggest either one of two states—with a simple drowsy condition, accelerated pulse and febrile temperature, along with active pupils, they suggest for the most part the beginning of the breaking-down process; or if they are seen, when the patient is developing the comatose state with lowered pulse and inactive pupils, they denote the absorption of a toxic agent. Similarly, convulsions are twofold in their significance; if general, and early in the disease, they are symbols of cerebral irritation, and do not indicate urgency; if they are seen, when other symptoms are thoroughly established and localised, and are followed by paralysis, they suggest pressure upon, or destruction of, highly important nerve centres, and mean that the diseased region is urgently crying out for relief. Further, if one or more fits occur after pus has been evacuated, it is very certain that either the cavity has not been drained properly, or that there is an independent collection.

Whatever the cause of the abscess may be, there is a primary inflammatory softening, accompanied for the most part by well-defined brain symptoms, but modified or obscured in the case of the breaking down of tubercle or gumma. Such softening may, in rare cases of traumatism and influenza, resolve itself, and the symptoms may disappear and operation become unnecessary, and, if the exploring needle is used at this stage, it may actually pass into the diseased part, but if no pus flows through it, the surgeon is apt to conclude that he has made a mistake in the *locale* of the disease, whereas his mistake is in having operated too early, as is sometimes evidenced by pus oozing from the puncture some time later.

To put it briefly, the *first* stage in this pyogenic process is that of irritation, and all that irritation implies; the *second* is that of pressure, and the *third* is pressure plus toxæmia. All the symptoms may not develop at the same rate, some one or more may remain stationary, as others become deepened or intensified. It is not for a moment suggested that the stages always admit of nice differentiation, but, excepting those rare cases, in which a person becomes suddenly maniacal through the severity of his headache, passes quickly into a state of coma, and, on operation, is found to be suffering from intracranial abscess, the sequence of events is very much as stated below.

The *first*, or *inflammatory*, stage may continue for days or weeks, and is generally represented by the following combination of symptoms, or by some of them: Continuous headache, heavy and general rather than severe and localised; if the latter, it is apt to be referred to the forehead, and is seldom paroxysmal. There is almost certainly vomiting, which has no relation to the ingestion of food, and is worse, if the trouble is cerebellar, but is as yet unassociated with nausea, and the bowels are constipated; the pulse is quick, and the temperature is febrile, or normal, but never subnormal. There is stupidity rather than stupor, drowsiness rather than sleep, and if the patient does sleep, he is easily wakened; the hearing is at fault on the diseased side, but he is by no means deaf, either to aërial or bone conduction of sound; he understands questions, but is irritable, and gives the enquirer to understand that he would rather be left alone. There is paresis of muscles rather than paralysis; there may be aphasia, alexia, amnesia, or agraphia, but this is unlikely. If the optic discs are examined, they will still be found normal, or only a slight papillitis will be observed; the pupils are equal and react to light and accommodation. If rigors have occurred, they have not been severe or followed by profuse perspiration; and, if with most of these symptoms present, there should be a diagnosis of basal meningitis, there can be little cause of surprise.

The *second* or *early purulent* stage, the pus being as yet inoffensive, is probably always short, lasting any time from a few hours to a few days, and this is the surgeon's golden opportunity. There is severe pain now, probably localised and associated with tenderness on pressure or percussion, vomiting with nausea is present, and, in all probability there is more of the latter than the former; the tongue is furred, and the breath is becoming fœtid; the pulse is below oftener than above the normal, and frequently weak and intermittent. As the disease advances, the temperature, in uncomplicated cases, tends to drop rather than rise, and it may be normal or just above or below that point. Respiration is slow, paralysis frequently marked. The patient may still be roused, or he may be quite unconscious, if he is conscious, the cerebration is very slow. Optic neuritis is probably present, but more obvious on the diseased side, on which side also the patient is likely to be quite deaf, except perhaps to bone conduction. The reflexes will be exaggerated, and, while the pupils may still respond, they are much more likely to be sluggish, or altogether inactive. While the patient still continues in this stage, some of the symptoms may be exceedingly grave.

The *third* or *toxæmic* stage is that in which the pus has become green in colour and highly offensive. Its effect upon the system is highly toxic, and increasingly so from hour to hour. If the surgeon now reaches pus, and establishes and keeps up the best possible drainage, the possibility of his pulling his patient through has not wholly disappeared, but it is infinitely less than it was in the preceding stage, most of the symptoms having become intensified, and the general condition being at low-water mark. The power of speech and thought will probably be gone, if there is not indeed deep coma. If the patient can express his feelings, he will speak of the nausea as beyond description, particularly in cerebellar abscess; the constipation may have given way to diarrhœa, and he may be passing urine and fæces involuntarily; the optic neuritis will probably be intense on both sides; the breathing will be slow, possibly stertorous, or of the Cheyne-Stokes character; the knee-jerks will be absent; and if, in addition to this grave condition, the pulse has dropped below 50 beats per minute, and the temperature is at or below 96° F.—the subnormal temperature must be considered to be of greater importance than the low pulse—his post-operation chances are still smaller; and if he has also contracted and insensitive, or dilated and unequal pupils, his chances are by no means improved.

Of particularly evil omen for recovery after operation, are convulsions occurring during the toxæmic stage, blindness without, or with little, optic neuritis; twitching of facial muscles in cerebellar abscess; and in cerebral abscess, combined low temperature and pulse, and contracted and insensitive pupils. With a person exhibiting several of the above serious symptoms, surgical procedure can only be undertaken as an extremely forlorn hope, and the surgeon who operates, and scores a success is a fortunate man.

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ON THE POSSIBILITY OF TWO WEST AFRICAN DISEASES ("AKATAMA") AND ("VONULO") BEING MALARIAL PAROXYSMAL NEUROSES.

By F. C. WELLMAN, M.D.

THE writer's attention has been directed to the article by Dr. Francis Hare on "Pathological Variations of Physiological Vaso-motor Action with Special Reference to the Malarial Paroxysmal Neuroses,"[1] and it has seemed that a sketch of two tropical affections, which would probably be placed by Dr. Hare with malarial vaso-motor disturbances, together with a brief statement of the results of some clinical and ætiological observations on the same, might be of interest to the readers of this Journal.

More than two years ago, I described a disease called "Akatama", which is common in the highlands of Portuguese West Africa,[2] and later, another well-known affection called "Vonulo" from the same region.[3] In studying both these conditions, several cases were examined with the express purpose of determining the relation of the diseases to malaria, and I would refer the reader to my original papers for the details of the investigations, only giving here the symptoms noted, and the ætiological conclusions adopted.

"AKATAMA."

This is an affection characterised by swelling erythema (which can be seen only in light-coloured natives), prickling sensations, burning, numbness, and sometimes excessive sweating. These are intensified by cold and damp, and relieved by the application of dry heat. The symptoms, just named, appear on the legs and arms, and occasionally on any part of the surface of the body. Aphasia, and other motor disturbances, are never seen. In severe cases the walk is peculiar, a tendency to curl the toes, as if walking on the heels and ends of the toes. I have seen severe cases, who could not walk at all in cold weather. The trouble cannot be confounded with any described disease. The following table shows the principal points of difference from other affections, which most resemble it :—

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[1] THE PRACTITIONER, August, 1905.

[2] *Journal of Tropical Medicine*, September 1, 1903, p. 267.

[3] *New York Medical Journal*, August 19, 1905, p. 377.

"AKATAMA."	STRACHAN'S DISEASE.[1]
Nerves of special sense (sight, hearing, etc.) not affected.	Nerves of special sense affected.
No wasting of muscles.	Wasting of muscles.
Vesicles along track of nerves with subsequent desquamation not seen.	Vesicles seen along track of nerves with subsequent desquamation.
No excoriation of muco-cutaneous orifices.	Excoriation of muco-cutaneous orifices seen.
Is probably a mild peripheral neuritis, or an angio-spastic neurosis.	Is a multiple neuritis.
	RAYNAUD'S DISEASE.[2]
"Dead finger," etc., never seen.	Such phenomena are constant symptoms.
Paleness of skin not a symptom.	Is a very common symptom.
No sensations of cold.	Is a prominent sign.
No cyanotic colour.	Often seen.
No clubbing of fingers, nor "Hippocratic nails."	Sometimes seen.
Never leads to gangrene.	Commonly leads to gangrene.
Comparatively slight pain.	Pain very severe.
No heart symptoms.	Sometimes seen.
	BERI-BERIC NEURITIS.
No pain in calves, lumbar region, etc., on pressure.	Pain on pressure at these points.
No heart symptoms.	Often prominent.
Œdema slight and transitory (not always present).	Very marked in the "wet" variety.
No atrophy.	In "dry" cases the most prominent symptom.
Reflexes normal and no ankle drop.	Reflexes not normal, and ankle drop often seen.
Limping walk as from pain, and changes from side to side. Not always present, and never ataxic.	Paralytic symptoms constant.
	MALARIAL NEURITIS AND NEUROSIS.
Parasites, enlarged spleens, etc., not found in greater *per cent.* of cases than in apparently healthy natives.	Associated with obvious malarial infection.

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[1] *Vide* THE PRACTITIONER, November, 1897, p. 477.

[2] Raynaud: *Thèse* (*Sydenham Soc. Trans.* by Barlow), also *Arch. Gén. de Méd.*, Vol. I., p. 5 *seq.*, 1874. For the relation of Raynaud's Disease to peripheral neuritis, *vide* Hochenegg, *Ueber symmetrische Gangran und locale Asphyxie*, p. 35 (Ref., Barlow, *op. cit.*).

Not amenable to quinine.	Relieved, or cured, by the exhibition of quinine.
	NEURITIS following dysentery.
"*Akatama*" occurs in individuals who have never had dysentery.	Case history positive.
	NEURITIS following diphtheria, typhoid fever, tuberculosis, etc.
These diseases are unknown among several tribes where "*Akatama*" is common.	Case history or clinical examination positive.
	SYPHILIS.
Seen where syphilis is absent.	Signs of syphilis present.
	LEAD POISONING, ALCOHOL POISONING, ETC.
Natives of region handle no lead. "*Akatama*" seen in natives, who cannot get rum to drink.	History of working in lead, and signs and history of alcoholism positive.
	LEPROSY.
Absence of *B. lepræ* from nasal discharges, etc.	*B. lepræ* can often be demonstrated.
No anæsthesia.	Often present.
No pale spots.	Not seldom seen.
No nodules.	Mark of nodular variety.
	ELEPHANTIASIS.
Swelling slight and transitory.	An important feature and permanent.
Part returns to normal size after attack.	Never entirely disappears.
No rise of temperature.	Fever accompanies the lymphangitis.
Skin shows no structural changes.	Structural changes marked.

"VONULO."

"*Vonulo*" is an affection of the bronchi and bronchioli, characterised by severe pains, sometimes of the shooting variety, in the chest, generally under the sternum. Occasionally accompanied by pain under one or both shoulder blades. Temperature and pulse are unchanged. Occasionally there is a feeling of constriction, but never marked dyspnœa. Aching, which is often worse at night. Rarely throbbing and tenderness. The only constant symptom is continual pain under the sternum. I give here its differentiation from other similar affections :—

"VONULO."	ASTHMA.
Dyspnœa generally not marked.	Marked dyspnœa.
Comes on gradually.	Attack sudden.
Never spasmodic.	Limited to spasmodic seizures.
Respiration slightly affected.	Forced expiration of air.
Little or no sputum.	Characteristic sputum.
No eosinophilia.	Eosinophilia.
Common in the district.	Very rare.
	PLASTIC PLEURISY.
No characteristic position.	Characteristic position assumed by patient.
No loss of respiratory motion.	Marked loss.
Friction sounds absent.	Sounds present.
No effusion.	Effusion.
	INTERSTITIAL PLEURISY.
Cough slight or absent.	Cough.
Hæmorrhages never seen.	Hæmorrhages.
Dyspnœa not marked.	Severe dyspnœa.
	CHRONIC BRONCHITIS.
Cough slight or absent.	Cough.
Little or no expectoration.	Expectoration.
No bronchiectasis.	Sometimes present.
	ANGINA PECTORIS.
Gradual.	Paroxysmal.
Severe exertion may intensify it, but emotion has no effect.	Supervenes on sudden exertion, or strong emotion.
Pain comparatively mild.	Terrible pain.
	HEPATITIS AND LIVER ABSCESS.
Not connected with dysentery.	Generally have history of dysentery.
No rigors or night-sweats.	Rigors and night-sweats.
Pain under shoulder may be absent.	Pain generally present.
Not uncommon.	Rare or absent.
	MALARIAL PAINS.
Per cent. of cases showing parasites in blood, and other signs of malaria not greater than in apparently healthy natures.	Associated with malarial infection.
Not amenable to quinine.	Amenable to quinine.
	MALARIAL SPLENITIS.
Numbers of cases occur in which the spleen is normal.	Spleen enlarged.

	ANKYLOSTOMIASIS.
Fæces examination negative in many cases.	Ova of worm seen in fæces.
	ENDEMIC HÆMOPTYSIS.
Examination of sputum always negative.	Hæmorrhages and ova of distomum present.
	TUBERCULOSIS, ACTINOMYCOSIS, HYDATIDS, ETC.
Sputum examination negative.	Sputum examination positive.

THE RELATION OF "AKATAMA" AND "VONULO" TO MALARIA.

In my papers referred to above, I have given *in extenso* my reasons for not connecting the two affections just sketched with malaria. I can present here but a *résumé* of my conclusions. In the case of "*akatama*," I have recently mentioned in a German periodical[1] a series of 31 cases, in which the average of malarial infections (as determined by spleen index, microscopical examination of blood, the *post-mortem* inspection of tissues, etc.) was slightly lower than that shown by statistics (used as controls), of about 500 blood and spleen examinations of other natures. It must be confessed, however, that I have not succeeded in establishing any other cause for the symptoms. But I would further point out here that there are, besides the negative statistics just mentioned, other reasons against connecting "*akatama*" with malaria. The geographical distribution of the two diseases constitutes one objection. "*Akatama*" seems to be confined to the Angola highlands of West Africa. The only suggestion of its possible existence, outside of Angola, that I have seen is from Dr. A. Balfour, of Khartoum.[2] Several medical men have kindly determined for me its absence from their districts, some of which are much more malarious than mine. Another point militating against its being a malarial vaso-motor paroxysmal neurosis is the fact that marked rigor is an extremely rare symptom of malaria in this district, one set of statistics showing only about one instance out of a hundred observed attacks of fever. Turning now to "*vonulo*," we find[3] that almost the same reasons obtain for considering it as distinct from malarial pains. It must again be admitted, however, that, as in the case of "*akatama*," no other cause has been demonstrated to account for the condition, although cultures have been obtained from the sputum, and careful examinations of the blood and excreta have been carried out in a series of cases. Accordingly, the possibility of these two little-known affections being malarial in origin must, of course, be always kept in mind, even though one has failed to demonstrate their connection, either with the other, or with a particular malarial infection. To me, the article of Dr. Hare, mentioned early in this paper, is very suggestive in this connection, as well as is a recent study by Dr. F. Schwyzer, of "Intermittent Angiospasm on the Basis of Chronic Malaria."[1] Yet I cannot (although I bear in mind the almost universal malarial infection in the inhabitants of tropical countries), on the basis of my studies so far made, definitely predicate malaria, or any other known cause, as the origin of the groups of symptoms respectively characterised by the natives of West Africa as "*akatama*" and "*vonulo*." Although I do not deny that malaria may very possibly be the factor in question, at the same time I do not yet see evidence that it is.

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[1] *Archiv. für Schiffs und Tropenhygiene*, Bd. X, Heft 3, 1906, p. 80. *Vid.* also *Revista Medica de S. Paulo*, May 15, 1906, p. 183.

[2] *Journal of Tropical Medicine*, April 15, 1904, p. 117.

[3] Vide *New York Medical Journal*, August 19, 1905, p. 378.

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[1] *Vide* Ref. in *Journ. Trop. Med.*, October 2, 1905, p. 295.

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ORTHOPÆDIC SURGERY.

By A. H. TUBBY, M.S., F.R.C.S.,

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ANKYLOSIS AND ITS OPERATIVE TREATMENT.

THE appearance of "Traitement des Ankyloses par la Résection Orthopédique," by Dr. Alphonse Huguier, marks a distinct advance in the treatment of the distressing deformities arising from joint disease. But the subject must be approached carefully. It is useless to make a new joint, if the stability of the limb is thereby destroyed, and it is equally futile to form a ne-arthrosis, if the result is to be that the limb renders less service to the individual than when it was ankylosed. It is, therefore, quite certain that there are conditions of ankylosis in good position, with which it is not permissible to interfere. It is also equally certain that there are some cases of vicious ankylosis, in which an operation can only be carried out so as to refix the joint in good position. Finally there are conditions under which it is either indispensable, or preferable for the increased utility of the limb, to re-establish the mobility of an articulation.

According to Ollier, three postulates exist for reconstituting an articulation. These are (1) the prevention of contact of the two surfaces of newly cut bone. A sufficiently wide piece must be removed, and the effects of reflex contraction of muscles in drawing the fragments together must be neutralised. So only can a sufficient space be left between the cut surfaces for the interposition of soft tissues; (2) the re-formation of new bone in the interfragmentary spaces must be prevented, and this is effected by taking away the periosteum corresponding to the portion of bone removed; (3) most important of all, in order to ensure permanent mobility, a serous bursa must be formed between the osseous fragments.

The first two requirements present no extreme difficulties, but the question as to which is the best kind of tissue for the production of a serous bursa needs careful consideration.

Dr. Huguier strongly advocates muscular tissue, and his view is supported by the experience and writings of J. B. Murphy, of Chicago, in his article on "Ankylosis, Arthroplasty, Clinical and Experimental" (*Journal of the American Medical Association*, May 20, 1905, p. 1573). Huguier details the experience of various operators with flaps of muscles, and aponeuroses with tendons and skin. The superiority of muscle over the other tissues mentioned is evident. It surrounds the joints, and, unlike the skin, it is aseptic from the first. Further, its vascular supply is exceedingly good, and it, therefore, bears partial detachment and transplantation extremely well. The objections, which apply to skin and to tendon-aponeuroses, are equally applicable to foreign bodies, whether absorbable or non-absorbable, interposed between joint surfaces.

Doubtless the selection of muscular tissue depends upon a recognition of the fact that the interposition of muscular fibre is one of the most potent causes of non-union of fracture. It appears that, after a time, muscle, covered by aponeurosis and fat, undergoes degeneration, and, when subjected to pressure, forms a hygroma, just as in some kinds of false joints, seen in the non-union of fracture of long bones, a pseudo-arthrosis is formed, the interior of which is lined with endothelium. Murphy has demonstrated[1] by experiments on animals, that such is the origin of the formation of these false joints, and has emphasised the utilisation of these pathological conditions in arthroplasty.

The indications for the formation of a ne-arthrosis depend upon the site and nature of the ankylosis, and the age of the patient. In the temporo-maxillary articulation, the results of an ankylosis are disastrous. The loss of the power of mastication, and the interference with breathing and phonation, result in the formation of that curious deformity of the face known to the German authors as "the bird-like visage." It also interferes with the development of the teeth, and is associated with rapid decay, shortly after eruption. In the sum total of the bad after-effects produced are cachexia and early decease. The effects may also suddenly become most serious. If these patients are seized with vomiting, there is a very strong probability of instant suffocation. So that a

[1] *Loc. cit.*

patient, suffering from ankylosis of the jaws should be strongly urged to submit to any procedure, which promises even partial relief only.

Ankylosis of the shoulder rarely calls for interference, and any operation is strongly contra-indicated, unless it can be clearly shown that the deltoid and the rotators of the humerus have escaped atrophy. If such is not the case, a useful member is likely to be transformed, by an indiscreet operation, into a flail-like limb. At the elbow, the question of the formation of a movable joint after ankylosis depends almost entirely upon the attitude of the limb when seen. If the elbow is fixed in full extension, or at an obtuse angle, the usefulness of the member is so much interfered with that intervention is called for. But, if the elbow is fixed at a right angle, in good position, then a policy of non-intervention is the best, especially if the patient is a mechanic, and requires to be possessed of stable limbs. Like the elbow, interference in the case of ankylosis of the thumb depends upon the attitude of the part, and the profession or occupation of the patient. But Dr. Huguier quotes a case of M. Nélaton, who re-established movement at the metacarpo-phalangeal articulation, and formed a new joint, which had those two qualities, pre-eminently to be desired in a ne-arthrosis, namely, mobility and stability.

At the hip, indications differ accordingly as to whether the ankylosis is uni- or bi-lateral. Unilateral ankylosis, with the limb in extension, that is to say, in the best possible position, makes a useful limb; but unilateral ankylosis, with vicious union, is extremely prejudicial. Many surgeons are content to do an Adams' or a Gant's operation, and straighten the limb, without in any way attempting to form a mobile articulation. But Huguier points out that operations for restoring the movement of the hip joint can be so carried out as to ensure mobility and stability.

In the case of double ankylosis of the hip, the condition of the patient is not so bad, if the limbs are in extension and parallel, but when distortion is present on both sides, and is dissimilar, the condition is truly pitiable. In these cases, reconstruction of the joint should be attempted, even at the risk of some loss of stability. There is a difference of opinion between surgeons, as to whether a mobile articulation should be made at one, or both joints. Volkmann, Langenbeck, and Ollier contented themselves with obtaining mobility on one side, and redressment into the extended position on the other side. Lorenz, on the contrary, thinks it advisable to obtain a double ne-arthrosis of such a character as to ensure sufficient mobility; but Nélaton prefers to operate on one side first, and, if the stability of the joint is sufficient, to repeat the proceeding on the opposite side; whereas, if the stability of the first operated upon is deficient, he contents himself with an ankylosis in the straight position on the other side. As to the knee, in spite of the favourable experiences of Fergusson and Annandale, the general opinion is that it is not advisable to seek to obtain mobility in an ankylosed knee by the interposition of muscle or other substance, and the same remark applies to the ankle.

The most important question, before undertaking an operation, is to consider carefully what pathological condition has been the cause of the ankylosis.

Experience shows that the liability to return of the ankylosis differs very much after these operations. There are some forms which never reappear, and others which may do so, in spite of a very large resection of bone and of the most careful after-treatment. Or, to put it more precisely, the reaction of the tissues after an operation depends very much upon the nature of the original disease. Certain ankylosing articular affections modify very little the properties of the tissues. Others, on the contrary, increase very much the osteogenetic activity of the bone cells, and impart to the fibrous tissues in the neighbourhood such a tendency to persistent contraction, extending over a very long period, that ankylosis often appears. The forms of inflammatory articular troubles, in which the resulting ankylosis is particularly difficult to deal with, are:—Gonorrhœal ankylosis, post-traumatic and infective arthritis after eruptive fevers, and the puerperal forms of ankylosis. In these types of cases, the danger of ankylosis is very great, and the possibility of recurrence of ankylosis, after the operation for the formation of a new joint, is so considerable that every precaution must be taken. Interposition of sufficient muscle and carefully regulated movements are most essential.

In the ankyloses consecutive to tubercular disease, the tissues have quite different characters from those found after the diseases mentioned above. Tubercular arthritis is characterised by comparatively little bone development, and by the formation of few bands of firm fibrous tissue, so that these patients are, when the tubercular disease has entirely subsided, very fit objects for operations designed to form mobile joints.

Another serious question, in the consideration of this subject, is the age of the patient. After thirty years of age, the power of the periosteal and bone cells to develop new bone becomes less active, and, therefore, re-ankylosis is less to be feared.

At the temporo-maxillary articulation, many forms of operation have been performed, but the one, which appears to be the most in favour, is resection of the condyle, either with or without resection of the coronoid process, and the interposition of a portion of the temporal or the masseter muscles. Another form of operation, which may be considered by the surgeon, is resection of the vertical ramus of the jaw, and inserting two strips of the masseter between the fragments. Before embarking on the operation, the surgeon must consider the advisability of performing a laryngotomy or tracheotomy, in view of the danger to the patient, if he should happen to vomit during the operation, or become embarrassed by a collection of blood in the pharynx when the ramus of the jaw is sawn through. For the operation on the condyle very many lines of incision have been used. The point is to avoid injuring the facial nerve trunk, or its branches, and Huguier recommends an incision commencing two fingers' breadth in front of the tragus, and three fingers' breadth above the zygoma. From that point the knife is carried vertically down to a little below the zygoma, and then backward nearly to the tragus. This has the advantage of avoiding all the important branches. By dissecting downwards, the ligaments of the articulation are exposed, the joint is opened, and the condyle is seen. The neck of the condyle is sawn, or chiselled through, great care being taken of the internal maxillary artery. The condyle is then carefully chiselled out of the glenoid cavity. The movements in doing this must be delicate, as otherwise its base will be perforated, and the cranial cavity opened. The condyle is now removed, and the remainder of the joint surfaces exposed. All periosteum in the neighbourhood should be removed, and all osteogenetic tissue in and about the glenoid cavity. It is also advisable in unilateral cases to resect the coronoid process. The zygoma is then removed for a distance of 1½ centimetres, and a tongue of muscular tissue, 4 centimetres long, and 1 centimetre wide, is then taken from the posterior fibres of the temporal muscles, in such a way that its upper free end can be turned backwards, and stitched to the parotid fascia, and, at the same time, cover the bared surface on the neck of the condyle. If the vertical ramus of the jaw is resected, muscular bands may be taken, either from the temporal or masseter, preferably the latter, but it is important that both fragments of the jaw should be covered with separate strips of muscle.

At the elbow, a longitudinal incision is made on its posterior surface; the periosteum is carefully dissected away and removed from all those parts of the bones which formerly corresponded with the articulation. The line of ankylosis is broken though: the ends of the bones are sawn off well above their former articular surfaces, and a piece of muscle interposed. Berger has used a strip from the anconeus, Delbet has used a portion of the brachialis anticus, and Quénu has grafted part of the aponeurosis of the triceps. After the operation, movement should begin in twelve or fifteen days.

The first interposition of muscle for ankylosis of the hip was made by M. Rochet of Lyons in 1895, and two other cases have since been done by the same surgeon. In the three cases, M. Rochet corrected a vicious attitude of the limb by osteotomies of the femur, either intra- or sub-trochanteric, and he interposed, between the fragments, bands of muscular tissue which he took from the psoas, or from the glutei. In the three cases, the results sought for were obtained. The new articulations remained mobile, but the range of movement was comparatively limited.

M. Nélaton, in 1899, performed a similar operation, using part of the rectus femoris. The hip recovered its movement, and the patient could do his work and walk well, although the gait was somewhat similar to that seen in congenital dislocation of the femur. In operating on the hip for this purpose, the osteotomy of the femur may be either transtrochanteric or within the limits of the joint. In the latter case, the osseous mass filling the cotyloid cavity is gradually gouged away until the cavity is fairly well defined, and the remainder of the upper part of the femur is fashioned so as to present a rounded end. A slip of muscle, 8 to 10 centimetres long, is then turned downwards and backwards from the rectus femoris. The free end is drawn in between the new cotyloid cavity and the upper end of the femur, and is fixed to the lower part of the cotyloid cavity. When the femur is ankylosed posteriorly and superiorly to the acetabulum, it is better to use a portion of the glutei muscles rather than the rectus. Dr. Huguier describes numerous cases in illustration of the procedure on several joints, and a careful perusal of the cases convinces us that this new form of operation holds out great possibilities.

In this connection, the articles by J. B. Murphy on Ankylosis and Arthroplasty, in the *Journal of the American Medical Association*, May 20 and 27, 1905, should be read. He operated most successfully on an ankylosed hip. The description is taken from the *Boston Medical and Surgical Journal*, May, 1905.

"One of the most brilliant contributions to Orthopædic Surgery comes from the hands of Dr. Murphy (*Transac. of Amer. Surgical Assoc.*, Vol. XXII., 1904; *Jour. Amer. Med. Assoc.*, May 20, 1905) of Chicago, who reported a successful case of operation for ankylosis of the hip joint treated by arthroplasty, or the interposition of a flap of fascia lata between the head and acetabulum which had become ankylosed after injury. A year after the operation there was complete function of the joint and normal motion. The method has been applied also at the knee joint by Dr. Murphy with success. Previous attempts of this method at the knee joint have met with but little success. These failures, in the opinion of Dr. Murphy, have been due to insufficient or defective exsection of the synovial membrane, capsule, and ligament; insufficient interposition of fat and aponeurosis, or of fat and muscle, between the separated bony surfaces, infection and sensitiveness to pain on motion after operation."

Dr. Murphy believes that, where osseous union has existed, it is important to disconnect the bones in as nearly their normal line of union as possible. This is best done by a narrow chisel. In the shoulder and hip, a chisel can be used to separate the bones so as to define the line of fracture in such a way as to secure the normal formation of head and cavity as far as possible.

All bony prominences should be removed. The soft parts should be thoroughly liberated. The muscular aponeurosis with fat, Dr. Murphy thinks, makes the best interposed tissue. If aponeurosis cannot be found, muscle and some fatty tissue should be substituted.

The development of a hygroma from pressure of fatty tissue should be the uniform result. If there is muscular contraction it should be overcome.

In the operation upon the hip, a U-shaped incision was made, 5 inches wide, with its base upwards, beginning 4 inches above the trochanter and extending downwards on either side for 2 inches. The incision was made through the skin, superficial fat, and fascia lata, and these tissues were dissected upward as one flap. A long curved needle threaded with heavy silk was passed around the base of the trochanter, beneath the muscles attached to it, and, by means of this carrier, a chain-saw was brought into position. The trochanter was sawn off and retracted upwards. The capsule of the joint was next incised and separated from the ilium all the way round. The attachment of the gluteus maximus was not disturbed.

The bony tissue filling the acetabulum was chiselled free from the latter by following round the margin with a carpenter's curved chisel an inch and a half in diameter. The chisel each time was driven in obliquely towards the acetabulum, the object of this being to make the new femoral head as nearly spherical as possible. When the chiselling process was completed, the new head was entirely separated from the sides of the acetabulum, and was attached only at the bottom by a narrow bridge of bone. This bridge was broken by forcible abduction of the limb, after which the head was dislocated from the cavity by external rotation of the thigh.

The shape and contour were almost exactly those of a normal femoral head. The bony projections were removed by a bone-cutting forceps. The fascia lata, which formed a part of the original U-shaped flap, was now dissected away from the subcutaneous fat tissue down to its base, where the vessels enter to supply it with blood. The flap of fascia was then drawn down under the head of the femur, and carefully fitted into the acetabular cavity to form a complete lining. The under surface of the flap half an inch from the edge was sutured to the joint capsule with interrupted catgut suture. The head of the femur was returned to its normal position. The free border of the flap was now sutured with interrupted catgut suture, and the capsule attached to the neck of the femur.

The flap was large enough to cover the head completely, and to come on to the neck without tension. The great trochanter was brought down to its normal position, and accurately secured. The skin flap was replaced, and the limb was put up in a plaster cast.

CHRONIC RIGIDITY OF THE SPINE.

To the *Medical Chronicle* of March, 1906, Dr. W. B. Warrington contributes an interesting and critical article on some cases of rigidity of the spine. These are often described as examples of arthritis deformans, or osteoarthritis. We are now well acquainted with the descriptions of Bechterew of stiffness and rigidity of the vertebral column as a special form of disease, and certain groups of cases are known by the name of Bechterew's disease. The leading features are immobility, or greatly diminished mobility, of the whole or part of the vertebral column, without pain on percussion, kyphosis of the upper dorsal region of the spine, with displacement of the head downwards and forwards, paresis and some atrophy of the muscles of the trunk and limb girdles. With these symptoms are associated marked signs of irritation of the spinal nerve roots, causing violent pains in the back, neck, and extremities, and an anaesthetic area in the region of supply of the upper dorsal nerves.

Some of the cases were hereditary, and some seemed to be due to traumatism.

The condition appears to be a meningo-myelitis, a somewhat different class of case from that known as the Marie-Strumpel type, or *spondylose rhizomyélique*, characterised by ankylosis of the spine, and of the articulations of the shoulder and pelvis girdles, but by escape of the small joints. In this type, the lower part of the spine is straightened out, while the upper part of the dorsal region shows kyphosis, with the head drooping forwards. The thighs are flexed, and the knees bent. Root pains are absent, but the rib joints are ankylosed, and the breathing is abdominal.

There is no doubt that formerly both these types of spinal mischief were included under the general heading of spondylitis deformans, but now we begin to recognise the necessity of regarding arthritis deformans of the spine as responsible for spondylitis deformans, and cases of the Bechterew and Marie Strumpel types as distinct entities. There is yet a great deal of work to be done on this subject, and careful clinical investigations are needed.

In this connection, the writings of Forrestier of Aix-les-Bains (*Archives Générales de Médecine*, 1901, page 56) are of distinct value.

SCOLIOSIS.

Any means of relief for this distressing condition are gladly welcomed, and a résumé of the Treatment of Scoliosis, by Dr. KLAPP ("System of Exercise," by Klapp, in the *Edinburgh Medical Journal*, July, 1906), is welcome. Dr. Klapp says that we are all familiar with the spinal contortionists of travelling shows, and the remarkable mobility possessed by their spinal columns. We must therefore aim, not only at the restoration of the normal function, but at the production of spinal specialists, or athletes, for it is only by a high development of musculature and mobility that any existing deformity can be overcome, or deformity prevented from occurring. Movements, which act more directly on the spinal column than trapeze and other gymnastic exercises, are required. Dr. Klapp has noted the lateral deviation, from side to side, of the spine in quadrupeds as they walk, the forward movements of each hind limb causing deviation, first to one side, then to the other. The exercises suggested by Dr. Klapp, therefore, consist of various movements of the four-footed type. The details of the exercises are too long to be quoted here, but a reference to Dr. Klapp's paper will show that there are great possibilities in this treatment, although no doubt the idea is at first somewhat repugnant.

Against the theory of these exercises, the present writer (A. H. T.) has seen three specimens of scoliosis appearing in four-footed animals.

At the Fourth General Congress of Orthopædic Surgeons (*Revue d'Orthopédie*, July 1, 1905, p. 370) the subject of scoliosis was carefully considered.

Lovett, of Boston, showed some experimental investigations into the movements of the lateral column in normal spines. The experiments were made upon fresh cadavera, with the pelvis immobilised, and controlled by observations on models. These experiments confirmed the statements, which have hitherto been made, to the effect that the lateral inclination of the spine is always accompanied by a rotation of the vertebral bodies with projection of the ribs on the concave side. This is attributed by Lovett to an instinctive attempt towards the re-establishment of the centre of gravity, interfered with by the lateral inclination.

In the report of the proceedings, it is stated that, in addition to this cause of the rotation, the shape of the articular facets and the structure of the vertebral column itself are factors which cannot be neglected.

Schultess, at the same meeting, presented clinical results in the study of 1,140 cases of lateral deviations of the spine. He classifies scoliosis thus: firstly, deformities due to primitive abnormalities of the vertebræ; secondly, due to an acquired anomaly of the vertebral column, that is, lesions of the skeleton, of the muscles, and of the nerves. He was able to show, through a series of tracings, the direction of the deviations according to the age of the patient, their relation, frequency, their tendency to increase or to remain stationary.

Schanz was able to find in scoliosis merely the resulting mechanical changes from an anomaly of superimposed weight on the spine. The questions of growth and age are only important in their relations to the excess of superimposed weight.

Riedinger accepted the theory of Scharz, but thought it was too rigid, and believed that there were variations.

SACROLUMBAR ARTICULATION.

Ludloff presented, before the meeting of the German Orthopædic Society, a paper on the anatomy of the sacrolumbar articulation, and showed that at the level of this articulation, and a little outside of it, a considerable hiatus exists. There is therefore here, a region of diminished resistance, and slight falls may determine lesions to the vertebral canal, from which persistent neuralgias may follow, the cause of which is often obscure.

SACRO-ILIAC ARTICULATION.

Goldthwait and Osgood presented an interesting paper (published in the *Boston Med. and Surgical Jour.*, May 1895) on the anatomy of the sacro-iliac articulation, claiming that laxity of these articulations, which is known to be developed in women at the time of childbirth, may also be developed in, the male, and give rise to symptoms of pain and disability, which may be treated by mechanical means of spinal fixation.

BIER'S CONGESTIVE TREATMENT FOR TUBERCULOSIS.

In the *Montreal Medical Journal*, a notice under the title of "Hyperamie als Heilmittel" is given of this form of treatment by F. S. Patch (*Montreal Medical Journal*, June, 1906).

It is well known that Bier was directed to the use of hyperæmia, as an agent against tuberculosis, by the observations of Farre and Travers, Louis and Friedrich, on the noticeable coincidence of pulmonary stenosis and pulmonary tuberculosis, and the assumption that the latter was due to the pulmonary anæmia resulting from the lesion. The contrary proposition was stated by Rokitansky, namely, the absence of tuberculosis, and especially of pulmonary tuberculosis, in obstructive heart lesions, where hyperæmia of the lungs is present.

After many trials, Bier contents himself with producing hyperæmia of the affected part for one hour daily, and the rules, which he has now laid down, are that the congestion must never cause pain, else it is falsely used, or the case is not a suitable one for this treatment. Again the congested part must never be cold, but there should be rather a local increase of temperature. He also notes that chronic œdema prevents the attainment of hyperæmia, and favours acute inflammation.

No distinction is made between closed and open tuberculosis, and they are both treated with *stauung Hyperämie*.

The contra-indications for the treatment are: (1) Commencing amyloid disease and the presence of tubercle in the viscera; (2) where the whole joint cavity is filled with pus or cold abscesses; (3) faulty position of the joint.

It seems that tuberculosis in the hand, foot, and elbow is more amenable to this form of treatment than in the shoulder or knee. Bier gives statistics of cases which he has treated by this method, from April 1, 1903, to August 1, 1904. In seventeen cases affecting the hand and wrist, in four, there were sinuses at the beginning, in five, abscesses had to be evacuated, but fifteen healed with free movement while two improved. The average length of treatment was twelve months. Eleven cases of elbow disease gave the following results:—Five of them had abscesses at the beginning and three later. In these eight cases the abscesses were opened during the treatment and they all healed. Absolutely normal movement was obtained in no case, but fairly free movement in all. The average length of treatment was nine months. Thirteen cases of tuberculosis of the foot came under observation, eight with sinuses, but all healed. Full movement was obtained in three cases, and satisfactory movements in the remainder. The average length of the treatment was ten months. Therefore healing ensued in 88 per cent. of the hand cases, 72·7 per cent. of the elbow, and 61·5 per cent. of the foot cases.

Bier has also had much success with *Saugtherapie* (the production of more or less vacuum by cupping glasses), so that many tuberculous cases with abscesses and sinuses are treated by this method.

EXTRA-ARTICULAR TUBERCULOSIS OF THE BONES.

In the *Albany Medical Annual* of August 1905, C. G. Cumston discusses this very important question. Tuberculosis of the bones, especially in children, begins as a deposit in the epiphysial ends. In rare cases there is a shower of multiple tubercular emboli, but much more frequently the disease is limited to one or more foci. As the foci increase in size, they spread steadily towards the articular surface of bone and periosteum, and the question arises as to why extension should take place in one direction or another, and why a tubercular abscess, developing in an epiphysis, should extend towards the joint, or the surface of the bone, and not down the shaft. Unfortunately, no definite answer can be given to this question. An extra-articular abscess has no distinct pathological character, but presents certain peculiarities in its relations. It is usually seated at one of the sides of the bone, and spreads out there. Occasionally, it is quite away from the joint, but very often has no direct connection with it. In some instances, it remains on the side of the epiphysis from which it has arisen, and does not extend beyond the growing line. At other times, it covers one entire side of a joint, envelops it, and is separated from the synovial membrane by the articular capsule only. If such an extra-articular abscess is opened, there may perhaps be no apparent communication with the bone, and even if the wall is scraped, and the periosteum explored with a sound, it may appear to be intact; yet, if one searches with extreme care, a small crack in the periosteum may be discovered, and a minute orifice may thus be detected, leading directly to the intra-osseous abscess cavity. In these cases of extra-articular tuberculosis of bones, and of para-articular abscess, it always appears as if the bone has increased in size. But this local hypertrophy is more apparent than real, and is due to thickening and induration of the soft parts, and not to hypertrophy of the bone. The neighbouring muscles are usually atrophied, but reflex contractions and shortening of them are not liable to occur. The neighbouring lymph glands are frequently enlarged. So long as the abscess remains purely extra-articular, but little change occurs within the synovial membrane, although, when pus approaches quite close to the articular cartilage, some serous fluid is poured out of the joint cavity. If the disease focus in the bone is situated at the point of insertion of one of the ligaments of the joint, then pain is produced when that ligament is tightened.

The question of how long a joint may remain free from disease, when an extra-articular tubercular focus exists, is interesting. Although nothing precise can be said, with the aid of statistics or otherwise, it is quite certain from observation that, at least in children, it may be for weeks, or even months, and during this time there is plenty of time at the surgeon's disposal to treat and cure the primary focus. In some situations, the localisation of tuberculosis in the epiphysis certainly does not result in very early participation and invasion of the joint. These situations are the lower end of the radius, the lower end of the fibula, and the great trochanter. There is a single point common to the lower ends of the radius and fibula, namely, the epiphysial cartilage is markedly above the level of the joint ; and with regard to the great trochanter, it is one of the only parts of the upper end of the femur, which can be invaded by tuberculosis, without early participation of the synovial membrane of the hip joint. Almost all other epiphyses are so covered by the synovial membrane that the involvement of one structure means imminent danger to the other.

With regard to the clinical characters of extra-articular tuberculosis, they may be considered in three, more or less distinct, stages. At first, the process develops insidiously and quietly, showing no symptom beyond pain, which is moderate, and can hardly be said to exist spontaneously. It is well marked after fatigue, but soon quiets down with rest. Local examination shows very little, but there is always a moderate decrease in the functional power of the limb, and this is not due to joint lesions, as one might be led to suppose, but arises from muscular atrophy. Cumston believes that muscular atrophy should be considered as the first sign of tuberculosis of the epiphyses. Examination of the diseased part will show the presence of a fixed, and very precise, painful spot, always the same, and exactly corresponding with the focus of disease. The sensitiveness, caused by even moderate pressure, is quite clearly defined, while palpation of the neighbouring parts is quite painless. Between this initial period, during which it is unusual for the patient to consult a surgeon, and the next period, which is characterised by the formation of a distinct swelling, there exists a more or less lengthy period of time, during which the lesion, which is still deeply seated, extends. Pain becomes more acute and superficial, although it retains the same character. Finally, the tumefaction of the bone appears, but it is still limited and hard, and another important symptom makes its appearance, namely, involvement of the lymphatics of the region.

In the second stage the pain becomes constant: it is spontaneous, and increases when the limb is moved. But an important point is this, that it is one particular movement which cause the pain, and this movement is the same in a given case There is no doubt that the cause of it is contraction of the muscles or ligaments, which take their insertion from the immediate neighbourhood of the diseased spot. Radiography is now of value, and shows that the swelling is confined to the soft parts. The tumefaction, which was at first perfectly circumscribed, now shows a tendency to extend.

This is the end of the second period, which culminates in suppuration of the tubercular deposit, when the skin frequently becomes involved. Now the third period is reached, when the extra-articular abscess is opened. It is of the greatest importance to recognise these periods of evolution of tuberculosis of bone, and if we were asked at which stage the danger to the joint was greatest, we should say at the end of the second stage. We do not know what factors determine irruption of pus into the joint, or the more favourable condition of escape of pus from the bone outside the joint, but every effort should be made to treat the case thoroughly before the joint has become involved, and there is no doubt that many joints have been saved by early recognition and adequate measures. So long as the disease is untreated it is a distinct danger to the joint.

The clinical characters of extra-articular tuberculosis have been insisted upon, and it now remains to distinguish it from intra-articular disease. A true arthritis from the start gives rise to rather sharp pain, which frequently takes on the character of neuralgia, with shooting pains at a distance from the joint. For example, the pain in the knee in coxitis, and the pain in the epigastrium in Pott's disease. Weakness appears in a very short time, and the movements become limited in all directions. Then contracture and shortening of muscles take place early, while extra-articular foci of disease are not accompanied by these signs. In joint disease, too, the lymphatic glands are often involved from the first, and, especially in the case of the hip joint, a distinct semi-solid thickening of the subcutaneous tissue takes place.

With reference to treament, the ideal plan is to cut down on the bone during the very early period of infection, when the one symptom pointing to trouble is fixed and localised pain. But too often this is not possible, and the matter only comes before the surgeon's notice when distinct intra-osseous thickening has occurred. By that time exposure of the diseased bone and thorough cleaning out of the cavity, followed by gouging, and the application of five per cent. chloride of zinc solution, is that form of treatment which is successful.

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Prize Essay.

THE RELATIVE VALUE AND THE SELECTION OF DISINFECTANTS.

BY G. H. BATE, L.S.A.,
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AT the present day, it is no easy matter to make a selection from among the host of disinfecting agents which are advocated by authorities and advertised by the proprietors. The general practitioner is confused by the circumstance that fresh agents are constantly being brought before his notice, the disinfectant value of which is vouched for by those who rank as authorities upon the subject ; and the individual, who endeavours to make his selection on merits (so far as he is capable of judging them from the published statements), finds "confusion worse confounded." It appears to one, who is outside the ranks of those who scientifically investigate the value of disinfecting agents, and who is endeavouring to keep himself *au courant* with a subject which is as interesting as it is important, that the present methods of gauging disinfectant values leave much to be desired ; and that it is impossible, from the published data relating to the agents mostly employed, to precisely arrange the order of their value, if regard is had to all the circumstances of employment in general practice. In order to maintain this argument, and at the same time to indicate the frequently overlooked conditions which affect the practical value of a disinfectant, it will be well to consider what are the requirements of an ideal disinfectant.

In the first place, the ideal disinfectant will be one which does not exhaust itself in an indiscriminate attack upon organic matter, living or dead ; one which will possess some specific power of killing the living germ without itself being too readily used up in the process. This desideratum will often exclude such agents as Chlorine and permanganate of potassium, and justify a preference for the acid perchloride of mercury, and such preparations as Izal and Cyllin.

The disinfecting agent should also be capable of achieving its object in a short time. It must, however, be borne in mind that, unless very great strengths are employed, a time limit must be allowed for any disinfecting agent to accomplish its object. It is for the most part a waste of good disinfectant, for instance, if it is added to a typhoid motion, and then the mixture is at once emptied down the watercloset basin. The proper practice is to allow a contact of at least 15 minutes, and, for that purpose, the bed-pan or chamber can be removed from the sick-room and placed in the watercloset apartment until the 15 to 30 minutes have elapsed. Another consideration often of importance is that the agent should not be poisonous; and it is further necessary that it should not be capable of injuring textile objects or receptacles. The former consideration will, unfortunately, often exclude from the sick-room the use of perchloride of mercury; while permanganate of potassium is absolutely excluded for the reason that, in the presence of organic matter, it would have to be employed at such strength that practically everything to which it would be exposed would be stained and disfigured.

The odour of a disinfectant is a matter which also demands some consideration. I am of opinion that there is an advantage in a slight odour, as sometimes it is necessary to disguise other odours in the sick-room. The odour of phenol is, however, most objectionable, and is so repellent to many, that one has often been asked to substitute some other agent for it. I have known the complaint made of headache or nausea from the odour of phenol, and I have, on several occasions, replaced it by preparations with a relatively pleasant and refreshing odour.

The power of a disinfectant to suffer no material loss upon exposure for several hours to the atmosphere is an important consideration when we bear in mind that, in practice, disinfectant solutions are frequently mixed, and then allowed to stand exposed in sick-rooms. There is no doubt that many disinfectants, which are very efficient when the bottle is freshly opened and the contents are first mixed, lose more or less of their power after a short exposure. Experiments have demonstrated the remarkable fact that this holds true, not only of disinfectants depending upon the action of free chlorine for their powers, the permanganate of potassium and other oxidising agents, and formic aldehyde solutions, but also of carbolic acid preparations. Perchloride of mercury and Izal stand such exposure without material reduction in strength.

There is an important consideration, which affects the use of disinfectants, and which is not always borne in mind. There is a tendency to use the disinfectants at the recognised strengths (say, the perchloride of mercury 1 in 1,000) in the disinfection of the contents of bed-pans, chambers, etc.; but it is obvious that, when such disinfectant is added, say, to an equal quantity of typhoid urine, the strength of the perchloride of mercury is no longer 1 in 1,000 but 1 in 2,000; and, therefore, whenever a disinfecting agent is to be added to a material which will dilute it to a considerable extent, it is necessary to provide against that by using the solution at double strength.

If disinfecting agents were judged by their compliance with the above requirements, it would not be a difficult matter to pick out those which approach closest to the ideal, assuming that we were familiar with the germicidal powers under all the conditions of practice; but it is here that we need more light. In the first place, it is known that different germs vary considerably in the ease or difficulty with which they are destroyed; it is also recognised that cultivations of even the same germs may vary somewhat in resistance, as they do in virulence; and it is a fact of fundamental importance to recognise in all experiments, which are designed to test the bactericidal powers of an agent under the conditions of actual practice, that the associated organic matter plays an important part in determining the strength at which a disinfectant must be employed; for the non-living organic matter, which shelters the bacteria (whether it be the sputum or dust in phthisis, or the faeces, urine, or dust in enteric fever, or the various textile materials infected by either disease), may either exhaust the disinfectant employed, or so weaken it as to render it ineffective upon the more resistant living germ.

Generally speaking, the experiments, on which we are asked to base our selection of a disinfectant, suffer from two defects :—They are either performed under very artificial conditions, upon what are practically naked germs; or the germs selected for experimentation are often those whose powers of resistance are known to be relatively low (as for instance the germs of plague and diphtheria). Judging from the trend of more recent work, these defects are now receiving recognition, and experimenters are turning their attention to standard methods, by the employment of which it should be possible to bring various disinfecting agents under a truly scientific comparison. It is precisely this which is needed; and, not until an acceptable standard method has been evolved, shall we emerge from the obscurity and confusion which at present baulk our efforts to arrive at true relative values. In the first place, it will be necessary to make the selection of a standard germ, and, for this purpose, to adopt either one of the most resistant specific organisms known, or a non-specific organism of at least equal resistance to the most resistant specific organisms; and, in the second place, a medium which (though it need not be fæces or sputum) must represent the maximum protective effect exerted by the organic matter with which the actual germs of disease are so generally associated. Nothing short of this will suffice, for the only safe course in practice is to assume that, in every case, the maximum difficulties may have to be encountered; and, even when the germ and its associated organic matter are known not to represent the maximum difficulty, the extra margin of safety, thus provided, is not likely to materially increase the cost and practical difficulty of disinfection, and it is a provision the importance of which will be appreciated generally.

A standard method was suggested two years ago by Messrs. Rideal and Walker, but Professors Kenwood and Hewlett, Mr. Meredith Wynter Blyth, and others have drawn attention to the defects in this method.

It is certain that a practical standard method for determining the relative values of various disinfectants under the conditions obtaining in practice is by no means easy of achievement, but the Rideal and Walker proposition was, that the standard method should be based upon the destruction of Bacillus typhosus in a broth culture, and that phenol should be the disinfectant selected as the standard for comparison. A carbolic acid co-efficient of any disinfecting agent is thereby attainable. But, as Professors Kenwood and Hewlett have pointed out, while Bacillus typhosus in fæces is hard to kill, in a trace of artificial medium it is so little resistant to disinfectants that it is dangerous to take such experiments as indicating the strength at which a disinfectant should be employed in common general practice, in order to kill the germ embedded in fæces or suspended in urine. Moreover, the experiments of Major Fowler, of the Royal Army Medical College, demonstrate that by such a test, which does not take into account the influence of the associated organic matter upon the disinfectant, permanganate of potassium, which is known to be of little value in the presence of organic matter, obtains a far higher coefficient than perchloride of mercury.

Moreover, some of the more practical experiments, which have been performed upon typhoid stools and urine by Klein and others, amply serve to demonstrate the need for the use of far higher strengths of the disinfecting agents when these are employed in the presence of organic matter, than when employed to destroy a germ in a trace of nutrient medium; and experiments have also demonstrated that, in fæces, the killing power of a disinfectant may vary sometimes to the extent of 25 per cent. with the nature and consistence of the fæces requiring disinfection. All these circumstances will serve to convince the practitioner that the only safe course to adopt is to allow a considerable margin for safety over and above the published results of the experiments hitherto performed.

The dejecta and discharges, collected from patients, may be disinfected by means of an acid solution of corrosive sublimate, which, in these cases should be used at double strength. The ordinary strength (1 in 1,000) can with advantage be made up in accordance with the recommendations of the Local Government Board, by mixing ½ oz. of corrosive sublimate, 1 oz. of hydrochloric acid, and 2 grains of aniline blue with 3 gallons of water. This is a cheap and powerful disinfectant, but it is one which suffers from two defects in practice. One of which is, that it has no power of killing the unpleasant odour of the dejecta, and the other, that it is poisonous. For this and other reasons, the best authorities appear to agree that such agents as Izal and Cyllin are to be preferred, at a strength of 2 to 5 per cent. I have carefully sifted the scientific testimony as to the efficiency of these two preparations. They have both stood the ordeal of most searching tests at the hands of recognised experimenters, with results which testify to very high and similar disinfecting values. Their slight but not unpleasant odour, their non-poisonous and good physical qualities as disinfectants, especially fit them for use in the sick-room. Where carbolic acid is not objected to, a 5 per cent. solution may be employed, and formic aldehyde solution is a useful agent to think of in this connection, at a strength of from 4 to 5 per cent.

Recent experiments have demonstrated the fact, that the frequent cleansing of the infected sick-room is a matter of great importance. More especially is this the case when cases of scarlet fever, diphtheria, and consumption are being nursed, and for the reason that the patient may, by coughing, spray the room with bacilli. Indeed it has been demonstrated that the fine spray, scattered by violent expiratory efforts, may remain suspended in the air for some time, before it is ultimately converted into dust. Flugge has shown that the minute drops of moisture sprayed by consumptives, even in speaking and sneezing, may disseminate tubercle bacilli to a distance of 4 feet from the patient. In these circumstances, the repeated cleansing of the surfaces of the room is a matter of special importance, and it is a desirable procedure in the case of any infectious disease. In this connection, mention must be made of the high disinfecting value of soap and water vigorously administered. This method is cheap and effectual for room surfaces, but it is more general, both in this country and abroad, to use the soap along with a disinfectant solution. Various combinations of soft soap with phenols and cresols are advocated on the Continent ; and those, who object to the odour given off, will find that, in practice, Izal and Sanitas mix well with soap, but that the use of soap with corrosive sublimate is difficult and unsatisfactory.

If the patient is likely to suffer injury from the effects of the evaporation of moisture when the floor, etc., is washed down, resource must be had to the removal of the dust daily by means of a damp rag, wrung out of Izal, or Cyllin 2 per cent., the rag to be subsequently burnt in the fire. In this connection, it may be mentioned that solutions of formic aldehyde should not be employed, as a rule, for cleansing the sick-rooms occupied by consumptives, as the vapours arising in the atmosphere often lead to the irritation of the sensitive respiratory tract of phthisical patients. A fire should be maintained, as a most valuable disinfecting agent, in every infectious sick-room, no matter what the time of year. It is quite easy by means of screens and by open windows to prevent a fire from unduly warming the room. It can then be used as the most efficient and rapid means of destroying the dust of the room, unfinished food brought to the sick-room, and pieces of rag, or Japanese paper (which may with advantage be used for wiping the nose and mouth and for collecting sputum in cases of consumption, diphtheria, and scarlet fever). At the same time, the open fire ensures a good draught of fresh air through the sick-room.

Phthisical sputum collected in spit-bottles is a material which, on account of its colloidal nature, is extremely difficult of disinfection. Experiments have demonstrated that the germs embedded in such material are capable of withstanding an exposure of half-an-hour when thoroughly mixed with a solution of 1 in 20 P.P. Phenol; and there can be little doubt that the attempt to disinfect sputum, by adding liquid disinfectant to the contents of a spit-cup, is a futile effort to achieve an object which can only be obtained by boiling or by burning. In order to facilitate the latter, the best arrangement that can be made is to provide the patient with the little cardboard spit-cases, which are now upon the market, and which can be subsequently put upon the fire.

Experiments testify to the very great difficulty of completely disinfecting the hands, and to the circumstance that soap and water (with the vigorous application of a nail-brush), followed by soakage for a minute or two in a 2 per thousand solution of the iodide of mercury, is one of the most efficient methods.

For instruments, which will not stand boiling for a quarter of an hour, soakage for one hour in 5 per cent. carbolic acid is efficient, and less likely to lead to any injury than if solutions of perchloride of mercury or formic aldehyde are employed.

The final disinfection of the sick-room, after the recovery or death of the patient, is now very generally undertaken by the sanitary authority, and, as the special difficulties of this disinfection are being more and more appreciated, the occasions on which the general practitioner takes the responsibility of advising that this disinfection should be carried out by the household, are getting very few indeed. To put the matter very briefly, it will be necessary to first disinfect the surfaces of the room, either by means of a fumigation method, or a spray method. With reference to the former, it may be said that, now that formic aldehyde has been proved so far superior in all respects to sulphurous acid, the continued use of the latter gas cannot be justified. Experiments have abundantly demonstrated that formic aldehyde is a gas which possesses the advantages over sulphurous acid of a greater germicidal power and a greater penetrating power, while at the same time it injures nothing which may be exposed to it in the room. The superior penetrating power is due to the fact that the specific gravity of the gas is practically identical with that of the atmosphere, whereas sulphurous acid is considerably heavier. Undue importance, however, has been attached, in many directions, to this matter, for in practice the superior penetrating effect of formic aldehyde counts for little, as it must be recognised that no gas or vapour employed in the fumigation of rooms, can, at the current temperature or pressure, penetrate sufficiently to remove the necessity for the subsequent steam disinfection of textile articles.

Authorities are in favour of Ligner's glyco-formal vaporising apparatus having furnished the best results in the hands of experimenters. The spraying method, however, is advocated by many, and generally consists in submitting all the surfaces of the room to a fine spray of either corrosive sublimate 1 in 1,000, or formic aldehyde 2 per cent. After this surface disinfection, the various textile articles, and indeed all articles, which may be penetrated by germs, require further disinfection; and there is no means of effecting this, except by soakage for at least an hour in a liquid disinfectant, by exposure to boiling water from a quarter to half an hour, or by allowing the sanitary authority to remove them for steam disinfection.

As to the relative value of the different apparatus in which steam is employed for the disinfection of textile articles and of the different kinds of steam so employed, there can be no doubt that saturated steam, at a temperature of about 115° C., employed in a chamber from which all the air can be moved prior to the introduction of the steam, is the quickest and most certain agent for penetrating and destroying the infection of textile articles. Many authorities hold the view that current steam at 103-104° C. is equally satisfactory in ordinary practice, and, more especially, if provision is made for first withdrawing air from the chamber. The latter provision, however, does not appear to ensure a sufficient margin of safety, and the former is therefore generally to be preferred. For leather, fur, and rubber goods, silk, books, brushes, combs, and feathers, which are injured by steam disinfection, the choice lies between a thorough fumigation, or spraying, by formic aldehyde and perchloride of mercury, respectively.

In conclusion, it may be asked whether our practice of disinfection achieves everything expected of it; and it is no easy matter to arrive at the actual truth. It is certain that the varying practice of disinfection throughout the country (both by local authorities and by individuals) appears to be attended with results which, so far as we can judge, present no very marked differences. But the difficulties of arriving at a just estimate of the efficiency of disinfecting methods are very considerable, for we have to recognise that the consequences of inefficiency will largely depend upon the nature of the infection, the probability of its retention, the nature of the subsequent exposure by different individuals, and the degree of their susceptibility; and it must, moreover, be borne in mind that the most susceptible material is often exhausted in the home by the primary outbreak of infection.

SOME FAMOUS QUACKS.

IV.—JOSHUA WARD.

ONE of the most famous quacks of the eighteenth century was Joshua Ward, or, as he was nicknamed from a claret mark on his cheek, "Spot Ward." His "Drop" and "Pill" were devoutly believed in by the fashionable world when George the Second was King, and brought their ingenious inventor considerable wealth and the advantages of a highly privileged position.

Unlike most quacks, Ward was a man of good family, being descended from the Wards of Wolverston Hall, Suffolk. He was born in 1685. Of his earlier years nothing is known with certainty. He is said to have begun life as a drysalter in Thames Street, London, in partnership with his brother William. He appears to have had some political ambition, and on January 27, 1716-17, he was returned to Parliament for Marlborough. His political career was short, for, on May 13, 1717, his name was erased by order of the House of Commons on the ground that he had not been properly returned. As he had not received a single vote, this conclusion is scarcely surprising, but the question how, in the circumstances, he got in remains a mystery as insoluble as the entrance of the apple into the dumpling seemed to the mind of George the Third. Before his Parliamentary misadventure, Ward had found it convenient to migrate to France, where he took refuge, first at St. Germains, and afterwards among the English Colony at Dunkirk. Why he left England so suddenly is not known; possibly he took some part in the rising of 1715, and this may account for his leaving the stormy sea of politics and confining his adventures to the quiet waters of medicine. William Wadd, in his *Nugae Chirurgicae*, says of him that "having failed in business, he applied himself to Chemistry, and in a propitious moment, hit on his famous Drop or Pill." This suggests bankruptcy as a possible explanation of his hegira from his native country.

During the whole of his stay in France, Ward supported himself by the sale of his nostrums, with which he professed to be able to cure every human ill.

But at this stage in his career, he seemed fated to get into collision with the civil power, for towards the close of his exile, he narrowly escaped imprisonment in the Bastille, and was in fact saved from that fate only by the good offices of John Page, afterwards M.P. for Chichester, and Secretary to the Treasury. Ward's gratitude for this gentleman's timely aid was such that at his death he confided to him the secret of his remedies.

Towards the end of the year 1733, Ward returned to England, having obtained a pardon from George II. He soon became famous, and partly by the accomplishment of some startling cures, partly by very extensive advertisement, he secured an enormous sale for his medicines. He was fortunate enough to win the good graces of the King himself by curing a dislocated thumb by a violent wrench. The monarch, at first displeased by this rough treatment, was afterwards very grateful for the completeness of the cure, and in gratitude assigned Ward an apartment in the Almonry, Whitehall, where he ministered to the poor at his Majesty's expense. He was also accorded the privilege of driving his carriage through St. James's Park. How highly this favour was prized at the time is shown by the story of the King's reply to some one having strong claims on his bounty who asked for it. "No," said the monarch, "I can't do that. But I will make you a peer of Ireland." His alleged success with the royal thumb would seem to show that Ward, in addition to the vending of his Pill and Drop, practised the ungentle art of bonesetting. Wadd, however, says that "an application he recommended for the King's hand established his reputation beyond competition."

However this may be, Ward's success with Royalty brought him into fashion. He found a most useful *vates sacer* at Court and in Society in General Churchill, and the "miraculous effects" of his pill on a servant maid were proclaimed by James Reynolds, Lord Chief Baron of the Exchequer. But those whom Warburton calls the "better vulgar" were not Ward's only patrons. Many people of real distinction placed themselves under his care. Henry Fielding, when dying, consulted him, and, in the *Voyage to Lisbon*, paid a high tribute to his sagacity and kindness. The passage is so interesting that we make no excuse for quoting it:

"I now became the patient of Dr. Ward, who wished I had taken his advice earlier. By his advice I was tapped, and fourteen quarts of water drawn from my belly. The sudden relaxation which this caused, added to my enervate, emaciated habit of body, so weakened me that within two days I was thought to be falling into the agonies of death. I was at the worst on that memorable day when the public lost Mr. Pelham. From that day I began slowly, as it were, to draw my feet out of the grave; till in two months' time I had again acquired some little degree of strength, but was again full of water. During this whole time I took Mr. Ward's medicines, which had seldom any perceptible operation. Those in particular of the diaphoretic kind, the working of which is thought to require a great strength of constitution to support, had so little effect on me, that Mr. Ward declared it was as vain to attempt sweating me as a deal board. In this situation I was tapped a second time. I had one quart of water less taken from me now than before; but I bore all the consequences of the operation much better. This I attributed greatly to a dose of laudanum prescribed by my surgeon. It first gave me the most delicious flow of spirits, and afterwards as comfortable a nap. . . . . Obligations to Mr. Ward I shall always confess; for I am convinced that he omitted no care in endeavouring to serve me, without any expectation or desire of fee or reward."

Fielding explains the failure of the treatment in his case as follows: "The powers of Mr. Ward's remedies want indeed no unfair puffs of mine to give them credit; and though this distemper of the dropsy stands, I believe, first in the list of those over which he is always certain of triumphing, yet, possibly, there might be something particular in my case capable of eluding that radical force which had healed so many thousands. The same distemper, in different constitutions, may possibly be attended with such different symptoms, that to find an infallible nostrum for the curing any one distemper in every patient may be almost as difficult as to find a panacea or the cure of all." The author of *Tom Jones* hated the medical profession as heartily as Molière, and loses no chance of gibing at doctors. He would not have been so indulgent in explaining any failures of the faculty. But he had that curious belief in unorthodox physic often seen in literary men, and he speaks as respectfully of Bishop Berkeley's tar water as he did of Ward's remedies, though in his own case both proved equally useless.

Gibbon, in his *Autobiography*, mentions Ward amongst those by whom his youth was "tortured or relieved." Lord Chesterfield was also one of his patrons. The second volume of Mathew Maty's *Memoirs* of that nobleman begins thus :—

"*Fog's Journal, Saturday, January* 17*th*, 1736.

"I very early took Mr. Ward's drop, notwithstanding the great discouragement it met with, in its infancy, from an honourable author, eminent for his political sagacity, who asserted it to be liquid popery and Jacobinism. I reaped great benefit from it, and recommended it to so many of my friends, that I question whether the author of that great specific is more obliged to any one man in the kingdom than myself, excepting one." It may be conjectured that the "one" here excepted was General Churchill.

But though Ward was the favourite of the fashionable world, he was so far unspoiled by their flattery as not to forget the poor, to whom he was always extremely generous. He bought three houses in Pimlico, near St. James's Park, which he turned into a hospital for his poorer patients; for their benefit also he took another house in the city, in Threadneedle Street. It became the fashion amongst the great ladies who consulted Ward to sit before his doors distributing his medicine to the huge crowds who resorted to him daily.

It need not be said that Ward had many enemies. Naturally, the medical profession was unfriendly, but he was also the mark for the satirists. One of them wrote :—

Of late, without the least pretence to skill,
Ward's grown a fam'd Physician by a Pill.

Charles Churchill, in the *Ghost* (Book 4), includes him in a sweeping denunciation of doctors:—

> Not such as those, who physic twirl,
> Full fraught with death, from every curl;
> Who prove, with all becoming state,
> Their voice to be the voice of Fate,
> Prepared with essence, drop or pill,
> To be another Ward or Hill,
> Before they can obtain their ends,
> To sign death-warrants for their friends,
> And talents vast as theirs employ,
> *Secundum artem* to destroy,
> Must pass (or laws their rage
> Before the chiefs of Warwick Lane restrain).

Ward was also bitterly attacked in the press, mainly it would seem, on political grounds. On November 28, 1734, a writer in the *Daily Courant* declared that the Pill and Drop were part of a new Papist plot. This sapient person's argument was that "from the Doctor himself being a Papist and distributing his Pills to the poor *gratis* by the hands of the Lady Gage, also a Papist, the Pill must be, beyond all doubt, a deep-laid plot to introduce Popery." Ward was so stung by some of these attacks that he foolishly went to law with one of his assailants; he lost his case and the emptiness of his pretensions was exposed. It was shown that, beyond a slight knowledge of pharmacy, he was destitute of medical knowledge; that his Pill and Drop were quite unsuitable for general use, being strong preparations of antimony, and that his remedies killed as many as they cured. It was hinted that they produced insanity, and there is a story in Turner's *Reprint of Miscellaneous Works and Memoirs of Chesterfield* that Queen Caroline once asked Churchill if it were true that Ward's medicines had made a man mad, and the poet answered: "Yes, madam—*Dr. Mead!*" Mead of course was a leader in the medical Israel of that day.

Horace Walpole, in a letter to Sir Horace Mann, dated January 20, 1760, writes:—"I don't know what to say about Ward's medicine, because the Cures he does in that complaint are performed by him in person. He rubs his hand with some preparation and holds it upon your forehead, from which several have found instant relief. If you please, I will consult him whether he will send you any preparation for it; but you must first send me the exact symptoms and circumstances of your disorder and constitution, for I would not for the world venture to transmit to you a blind remedy for any unexamined complaint."

But no exposures lessened the public faith in Ward. Even when, in 1748, an Apothecaries' Act was introduced into Parliament to restrain unlicensed persons from compounding medicines, a clause was inserted specially exempting him by name from the restrictions imposed. Doubtless encouraged by this flattering expression of belief in his power, Ward in later life enlarged the number of his nostrums, adding to his pharmacopœia, amongst other things, a very injurious eye-wash. His pills were also elaborated into three varieties, blue, red, and purple, all containing antimony, and two of them arsenic. He made attempts to manufacture porcelain and saltpetre, and was the first to bring into notice in England the method of preparing sulphuric acid by burning the sulphur with saltpetre. He took out a patent for his invention on June 23, 1749. The manufacture was carried on with the greatest secrecy, first at Twickenham, and afterwards at Richmond. Brande, in his *Manual of Chemistry*, says that the stench from Ward's works caused intense annoyance to the residents in these districts.

Ward died at Whitehall, on November 21, 1761. He left a considerable fortune, the bulk of which he bequeathed to his great-niece, Rebecca, the daughter of Knox Ward, Clarencieux King of Arms, and to his sisters, Margaret Gansel and Ann Manley. The sons of Knox Ward, Ralph and Thomas, are also mentioned in the will, which, dated March 1, 1760, was printed in the *Gentleman's Magazine* in 1762. In it he desired to be buried in front of the altar in Westminster Abbey, or "as near to the altar as might be."

It has already been mentioned that the secrets of his medicine were bequeathed to John Page, who had succoured him in France. Page published them under the title of *Receipts for Preparing and Compounding the Principal Medicines made use of by the late Mr. Ward* (London, 1763). He arranged that the profits from the sale of the medicines should be divided between the Asylum for Female Orphans and the Magdalen; he placed the charity under the charge of Sir

John Fielding. At first the sale brought in a considerable revenue, but, deprived of the advertisement of Ward's personality, and robbed of the mystery that had always surrounded them in his lifetime, they soon fell into disuse.

That Ward was not devoid of shrewdness may be gathered from what is said about him by Wadd, who as an orthodox practitioner, and surgeon extraordinary to George IV, may be regarded as an impartial authority. He speaks of his practice as "bold, and generally speaking successful in a class of diseases which previously had been a stumbling-block to the practitioners of physic. He met with great opposition; but he silenced all his adversaries by a long list of hopeless cases abandoned by the faculty, fortunately treated by himself." It cannot be questioned that rational medicine has learned much from quacks. The true principle of progress in the healing art is to try all things with an open mind and hold fast to that which is found to be good.

Reviews of Books.

Lectures on Tropical Diseases. By Sir PATRICK MANSON, K.C.M.G., M.D., LL.D. (Aber.); F.R.C.P. (Lond.); F.R.S., Hon. D.Sc. (Oxon.); Medical Adviser to the Colonial Office; Lecturer, London School of Tropical Medicine, St. George's Hospital Medical School, etc., etc. Pp. 130. London: Archibald Constable & Co., Ltd. 7s. 6d. net.

THESE are the "Lane Lectures," which were delivered at the Cooper Medical College, San Francisco, U.S.A., in August 1905. The book consists of ten chapters, dealing with the principles determining the geographical distribution of tropical diseases, ankylostomiasis, dracontiasis, bilharziosis, filariasis, malaria, trypanosomiasis, febrile tropical splenomegaly, and concludes with a discussion of the diagnosis of tropical fevers, the treatment of fevers and fluxes; also the general problems of tropical medicine, including the possible introduction of the yellow fever parasite into Asia, by means of steamers passing through a newly constructed Panama Canal.

The chapter on ankylostomiasis is exceedingly luminous and instructive, and should set at rest any controversy as to the way in which this disease is propagated. We have pleasure in calling especial attention to the accounts given of the life-history of the Guinea-worm, and to that of the parasites which cause bilharzia, filaria, and malaria. They are full of the most interesting information, and read almost like a romance; indeed the general style of the lectures is altogether attractive and entertaining, and at the same time, the matter is most instructive. The book is well illustrated, and cannot fail to be highly appreciated, not only by those interested in tropical diseases, but also by those who desire to keep themselves abreast of modern research.

Pathology, General and Special, for Students of Medicine. BY R. TANNER HEWLETT, M.D., F.R.C.P., D.P.H., Professor of General Pathology and Bacteriology in King's College, London; Lecturer on Bacteriology London School of Tropical Medicine. Pp. 540. London: J. & A. Churchill. 10s. 6d. net.

THERE are many excellent text-books upon the very extensive subject of pathology, and of some of the more important of these Professor Hewlett made free use when writing this excellent little book. Medical students are often at a loss to know what book they should read in pathology, as they usually find that they have insufficient time for the large text-books. After carefully looking through Dr. Hewlett's Pathology, we have come to the conclusion that it is just the book that will meet the requirements of all senior medical students, who may be reading for their final examinations, and we congratulate Professor Hewlett upon its excellence. The book is well arranged, and is embellished with some twenty-t plates, all of which have been carefully executed.

Æquanimitas, with other Addresses to Medical Students, Nurses, and Practitioners of Medicine. By William Osler, M.D., F.R.S., Regius Professor of Medicine, Oxford. London: H. K. Lewis.

As indicated above, the title of this book is that of the first address, which occupies nine pages only. The other addresses have relation to different topics, all of which are of vital interest, not only to the medical profession, but also to the public at large. They are all couched in terms clear, illuminating, and not unfrequently rising to a very high degree of eloquence, and may consequently be commended without hesitation to all classes of intelligent readers. We can, of course, refer only to a very limited number of these valuable addresses.

Professor Osler translates "æquanimitas" as *imperturbability*, and explains it as meaning coolness and presence of mind in all circumstances, calmness amid storm, clearness of judgment in moments of grave peril, immobility, impassiveness, and says that, as it is a bodily endowment, some, owing to congenital defects, may never be able to acquire it. We venture to think that it is a quality without which no practitioner can succeed in his profession, and that it can be acquired by all who resolutely set before themselves the high ideals of their profession, and that it is the outcome of a confidence derived from patient study and perfect skill.

The Address entitled "Teacher and Student" may be fairly compared with the noblest and most faithful ordination charge given by a Bishop to a company of newly ordained ministers of the gospel. It teems with advice and instruction of the highest order, and cannot fail to bear fruit in the minds of all who read it.

For playful badinage, and common-sense suggestions, we can unhesitatingly commend to all whom it may concern, and the constituency is a wide one, the Address on "Nurse and Patient," while we are of opinion that no one can afford to omit the valuable chapter on "Medicine in the Nineteenth Century."

In view of the efforts which are being made in the direction of improving the general system of medical graduation, the Address on "The Hospital as a College" is timely and worthy of careful consideration.

Much may be said in favour of those who have passed the "Fixed Period" of life retiring from active work in favour of their younger colleagues, but we venture to think that no hard-and-fast line can be drawn. Many men, whom we could name in literature, science, and politics, long past the meridian of age, are still a *tour de force* in their respective spheres, and will be so, let us hope, for years to come. So much depends upon constitution and previous history. But we forbear. To all we would, without reserve or hesitation, say of Professor Osler's book, read, mark, learn, and inwardly digest.

Psychiatry: A Text Book for Students and Physicians. By Stewart Paton, M.D. Pp. xii + 618. Philadelphia and London: Lippincott & Co. 18s. net.

We are informed that the object of this book is "to call attention to that aspect of the subject, which is in accord with the results of observations, as they are conducted to-day at the bedside and in the laboratory; and while presenting the different views of leading authorities in a manner readily to be comprehended by students of this important branch of medicine, to stimulate to greater activity the interest in the investigation of problems in the solution of which will be found the means of increasing the brain-power of the nation."

Upon the whole, the author is to be congratulated on the success of his effort. He has taken pains to present a readable and interesting account of the present state of the subject, and we believe that his book will be of real value to the intending student; albeit the frequent presentation of "different views of leading authorities" with which we meet, is a rather serious drawback, hiding from us, as it does, almost completely the personality of the author.

The first seven chapters, comprising nearly half of the book, are devoted to the nature of neuro-pathological conditions, to the general symptoms of insanity, and to the treatment and mode of examination of patients. The various mental diseases are then classified and described in a scheme of thirteen groups. We have no desire to criticise this scheme of classification; in the present state of psychiatry, there must be well-nigh *quot homines tot sententiæ.* We prefer to direct attention to another aspect of the subject.

"It would be superfluous," says Dr. Paton, "to emphasize the necessity of far more active coöperation in America between psychologist and psychiatrist were there not abundant evidence of the lack of communism of interests." "Psycho-pathology does not begin and end merely by measuring the promptness of reactions; its chief service has been in an analysis of the attention, in the study of the character of the individual reactions, of the anomalies of connected thought, and in investigating the sharpness and correctness, under varying conditions, of individual judgment." There is, indeed, good reason to lament that in this country, as well as in America, so little intimacy exists between normal psychology and psychiatry. The psychiatrist pursues his way, heedless—nay, ignorant—of the methods and possibilities of experimental psychology: the psychologist is often too little versed in knowledge of the abnormal states of mind. In point of fact, Dr. Paton himself reveals a lack of familiarity with psychological lines of thought. But he, at least, takes up a correct attitude as to the position of psychiatry in relation to neurology and psychology.

Unconscious Therapeutics, or the Personality of the Physician. By ALFRED T. SCHOFIELD, M.D., M.R.C.S., &c., Hon. Physician to the Friedenheim Hospital, author of the Unconscious Mind, the Force of Mind, &c. Pp. xii + 317. London: J. & A. Churchill. 5s. net.

THE title of this book scarcely indicates its object and scope, for "Unconscious Therapeutics" or the influence of the "Unconscious Mind" in relation to certain diseases applies more appropriately to the patient than it does to the physician. At the same time we are of opinion that Dr. Schofield has succeeded in making out a very strong case for a more systematic study of the influence of the mind in unconsciously causing and healing diseases, especially those of the nervous system. He very clearly shows that the cures, claimed by the vendors of patent medicines and by faith-healers, are really attributable to Unconscious Therapeutics, and cites cases in which diseases of long duration were cured in hospital, almost instantaneously, by judicious medical treatment. Two chapters in the books, those on *Vis Medicatrix Naturæ* and *The Personality of the Physician*, are especially worthy of consideration. In the latter, the effect of the "Unconscious Mind" of the Physician upon his patient, for weal or woe, is indisputably shown, and great stress is laid upon the need for the suitable training of Medical Practitioners in the methods which should be adopted in dealing with patients, and in studying their mental characteristics. In some Theological Colleges in this country there is a Professor of *Pastoral* Theology whose duty is described by his title. It might be well if a Professor with somewhat analogous duty were attached to our Medical Colleges. We have no hesitation in saying that Dr. Schofield's book deserves very careful consideration.

Sanatoria for Consumptives. A critical and detailed description, together with an exposition of the Open Air or Hygienic Treatment of Phthisis. By F. RUFENACHT WALTERS, M.D., M.R.C.P., F.R.C.S., Late Physician to the Mount Vernon Hospital for Consumption and Diseases of the Chest. London: Swan, Sonnenschein & Co., Ltd. New York: E. P. Dutton & Co. 12s. 6d. net.

As indicated by its title, this work is a sort of Directory of existing Sanatoria for Consumptive Patients, and Dr. Walters is to be congratulated on the very praiseworthy manner in which he has completed a very laborious task. The information afforded in the book has been obtained from the most trustworthy sources, and should prove of the utmost possible value to medical men, upon whom the responsibility of recommending sanatorium treatment to their patients devolves. The value of the book is considerably enhanced by a large number of beautiful illustrations, and very useful plans.

During the present month Mr. H. K. Lewis will establish his circulating library in more convenient quarters adjoining the old premises at 136, Gower Street. The old shop will be thrown into the new one, and will be utilised chiefly as the second-hand department. The new premises face Gower Street and Gower Place, and will afford greater convenience to customers to inspect the latest books in all branches. Subscribers will be able to examine the books on the shelves when making exchanges, and convenience for writing or making extracts will be provided, the idea being to make this department a reading room for subscribers, especially those residing in the suburbs or the provinces. The telephone in this room will also be free for subscribers' use, so that clients from the country may arrange appointments conveniently. Lavatory accommodation will also be provided, for the necessary "wash and brush up" after a journey, or a prolonged inspection of books.

The premises, which will still be known as 136, Gower Street, are near the Metropolitan Station, and are within easy access of various railways and omnibus routes. We congratulate Mr. Lewis on his enterprise.

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Notes by the Way.

The Influenza Number.

THE Editor wishes to express his regret that, owing to an unfortunate misunderstanding, advance pages of the January number were sent to the Press, and begs leave to apologise to the distinguished contributors for the mistake. The Editor need hardly say that he strongly disapproves of the way in which the names of the authors were displayed by the newspapers, for which he was in no way responsible.

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Cancer Cranks.

THE "cancer curer" we have always had with us; we are now suffering from a severe visitation of the "cancer crank." By this we mean the highly scientific person who, knowing nothing of practical medicine, and indeed often holding it in the contempt naturally born of ignorance, attacks the problem of cancer from a purely biological standpoint, and having satisfied himself that it is all a case of the anomalous behaviour of certain cells, forthwith publishes this as an epoch-making discovery. He then finds something which he persuades himself will eliminate the peccant cell and reduce the anarchy of disease to the order of health. It is all very plausible, but as cancer cannot be cured by a theory, however seductive, it is found necessary to support it by a few facts. These can generally be found without much difficulty. The possession of a medical degree, though it may to a certain extent be a test of knowledge, does not necessarily confer wisdom. Men whose academic honours almost exhaust the alphabet are often utterly lacking in judgment and critical power. They allow themselves to be carried away by the wind of every new doctrine, and do infinite harm by premature reports of the virtues of the latest therapeutic novelty. These men are especially apt to be led astray by the cancer crank. They publish reports of a few cases in which the disease seems to have been alleviated for a few weeks. Then the journalist, ever in quest of a new sensation, gets hold of these, and straightway up flares the rocket proclaiming the new discovery. When in a short time the dead stick comes down, nobody takes much heed. The cancer crank has had his advertisement ; the newspaper man says with the miser in Horace—

> At mihi plaudo
> Ipse domi nummos dum contemplor in arca ;

and the public forgets all about the matter. There is, it is true, one person who suffers—the patient who has been deluded with false hopes. What is play to the cranks and the scribblers is death to him. It may in charity be hoped that the irresponsible scribes, who publish such statements, do not realise the harm they do ; but they cannot be acquitted of a carelessness which is almost criminal. It is vain to appeal to the editors of the papers which live by sensationalism not to raise false hopes in sufferers who lie under the shadow of a cruel doom. But that men who are members of the medical profession should be found proclaiming that cancer has been conquered when the facts do not warrant the belief that it is even scotched, is a misfortune as far as the public are concerned, and to the profession something of a reproach. By all means let trypsin be fully tried, but to vaunt it as a "cure," before anything at all like a cure has been achieved, is a crime against mankind.

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An Eminent Physician's Advice.

To those journals, which have sufficient humanity to feel the wrong done by the awakening of delusive hopes in unhappy sufferers, we commend some wise words of Sir Thomas Barlow, which we found quoted in the *St. Louis Medical Review*, of October 13. When he was on the point of starting from New York, homeward bound, after the meeting of the British Medical Association at Toronto, he is said to have delivered himself as follows :—

"Undue alarm may be created among the ignorant and false hopes aroused. I have seen countless publications concerning cancer and the probability of its cure. Unscrupulous practitioners have asserted that the germ of the disease has been discovered, and with the discovery the production of an antitoxin which would destroy it as we destroy the germ of diphtheria.

"The truth is that the medical profession is no nearer the cause or the cure of cancer to-day than it was a hundred years ago. We still are delving and hoping. We are making progress slowly, but we are not yet within sight of the goal. Assertions have been made by irresponsible physicians that the germ has been isolated. The statement is false. The germ of cancer is yet to be discovered."

We may add that the discovery either of the germ or of the remedy can only be hindered by the premature pæans of frothy enthusiasts.

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The Fishy Theory of Leprosy.

THE theory of leprosy, which is advocated with such ability and persistence by Mr. Jonathan Hutchinson in the *Times* and elsewhere, was not of course originated by that distinguished surgeon. It is the oldest theory of all, and had acquired an ancient and fishlike smell that made it stink in the nostrils of the profession till he quickened it into an artificial freshness. We do not propose to discuss the question here, but as Mr. Hutchinson has repeatedly attacked the method of dealing with leprosy adopted in South Africa, we think it right to call attention to a debate on the subject which took place at the South African Medical Congress held last October. The following account is condensed from a report published in the *South African Medical Record* of October 10, 1906. Dr. Macfarlane, of Leribe, read a paper on "Leprosy in Northern Basutoland," in which he said that, although leprosy was fairly rife in that district, the people were absolutely non-fish-eaters, save only occasional sardines, which were also freely consumed by the Europeans. Dr. Dru-Drury expressed a hope that the paper would be published in Europe, where it might do much good. He had a painful recollection of hearing Mr. Hutchinson astonishing the Swansea meeting of the British Medical Association by insisting in a theory, which no authority on the subject upheld, and most unjustifiably attacking the South African methods of dealing with lepers. Only such steady work as Dr. Macfarlane's would stop a mischievous agitation. Dr. Gordon Grant stated that he had seen much leprosy in the Southern Sahara. The people were fish-eaters to some extent, but the fish was not salted, salt being a rare and expensive commodity. Segregation had been practised as far back as records went. One city he knew, with 1½ million inhabitants, had 1,850 lepers, all segregated. By the Islamic law segregation was compulsory. Under British influence the power of the chief to enforce the law had in some places been prevented, but wherever possible the chiefs carried segregation out, although the residents now compelled provision to be made for the lepers, instead of their being turned out of the city to starve as formerly. Cases of infection in families were by no means rare. Dr. Symington related his experience of nine months in Western Pondoland, where he saw several cases, mostly in blood relations, of patients who had been segregated. The people were not fish-eaters, fish being very rarely eaten, and then fresh. Dr. Wall said that the Transkeian natives regarded fish-eaters with the utmost contempt. Even the coast natives would eat fish only when compelled by hunger. Dr. Swift had seen 600 cases on Robben Island, but never anything bearing out the fish theory. There was often good evidence of contact, and sometimes of heredity. The question of how the organism gained entrance was a difficult one, but the fact of ulceration of the nasal mucosa being often noticed in early anæsthetic cases lent colour to the theory that the entrance was by way of the nasal passages. Col. Porter said he had seen a good deal of leprosy. His experience on the West Coast of Africa and in India had almost made him a convert to Mr. Hutchinson's theories, although in both these countries, salt being expensive, very little salt fish was eaten, the staple article in Ashantee being what is called "stink fish." But what he had seen and heard in South Africa had quite changed his opinions. Most of the members of the Leprosy Commission, whom he had met on their return from America, were of opinion that the disease was due to contagion or heredity. It was a most important question, and the South African Governments might surely subsidise research directed to the solution of the problem.

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The Noise Nuisance.

THE Chief Commissioner of Police is now wrestling with a difficult problem, namely, how to measure noise accurately so as to determine what is "undue" noise. A committee, of which Lord Rayleigh is a member, has been appointed to examine the question. Doubtless the multitudinous traffic of our streets cannot be carried on without noise, but we think some part of it at least is preventable. Is it necessary, for instance, that a railway station should be like an *inferno* in which the ears of travellers are rent as by the shrieks of damned souls? If constant whistling be necessary, could it not be made less excruciatingly shrill? So long ago as in 1890, at the Annual Meeting of the British Medical Association held at Birmingham, Dr. Thomas Barr called attention to the injurious effects of railway whistling on the ears. These he attributed to the very high pitch of the whistles; to their sudden and unexpected character; to their frequency in a covered or confined space such as a railway station or tunnel; and to their frequent proximity to the ears. He said it had been shown by Moos that, owing to their exposure to these influences, drivers and stokers suffered in an especial degree from defective hearing, even to such an extent as to endanger the safety of railway traffic. Dr. Barr added that he was convinced from his own experience that a not inconsiderable amount of ear disease was produced or aggravated by railway whistles. After some discussion, a resolution was passed that the attention of the leading railway companies should be drawn to the subject. What came of this? Probably nothing more than a perfunctory reply of the usual official type. Certainly nothing has been done to abate what is an abominable nuisance which could be mitigated by reducing the whistling to what is strictly necessary and using whistles of lower pitch and more musical quality, such as the "organ whistle" which was introduced on the Caledonian Railway Company's lines.

There is, we believe, a society in London which professes to make the abatement of this evil the object of its existence, but we have not observed that its labours have been crowned with any appreciable success. A "Society for the Suppression of Unnecessary Noise" has recently been founded in New York. All this shows that people are becoming restive under an infliction which is becoming more and more a nuisance and a danger.

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Place aux Dames.

ONE by one the strongholds of medicine have fallen to the conquering blast of the feminine trumpet. Women can now get medical degrees at all Universities except Oxford and Cambridge, over which there still hangs the shadow of an impenetrable conservatism; and they are admitted by all corporations except the London College of Physicians and the English College of Surgeons. The ladies, however, have not fought so strenuous a fight against the forces of prejudice without learning how to carry the warfare to a victorious issue. They are now summoning the fortress of surgery in Lincoln's Inn Fields to surrender. They urge that since they last battered at its doors in 1895, the entry of women into the medical profession has steadily increased, and the total number now on the *Medical Register* is over 750. Of these more than 400 have been students of the London School of Medicine for Women, and it is pointed out that to students in England the opportunity of obtaining the qualifications of the English Colleges would be especially valuable. That women have fully established their claim to the freedom of the City of Medicine is shown by the numbers who have won the difficult distinction of a London degree, and by the number of appointments which they hold on the staffs of hospitals and under public bodies throughout the country. Their work in India is recognised as helping the consolidation of the British Empire, and it is for the most part work which could not be done by men. We think the ladies have made out a good case for themselves, but we cannot feel much confidence that they will succeed in forcing their way into the sacred precincts of the College of Surgeons.

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Women and the Medical Profession.

THE prejudice, with which the bulk of the profession once regarded the question of the admission of women, has to a large extent died out. How far this may be due to the fact that they have not proved formidable rivals to medical men, need not be discussed. We will content ourselves with the statement of the fact. We do not know of one who has added anything of the highest value to the sum of knowledge; nor do we remember any who have been brilliantly successful in practice. A handful earn considerable incomes, and a good many are earning a comfortable livelihood. For the majority we fear the life of a practitioner of medicine is one of struggle. It may be said that, on the whole, in proportion to their numbers, they are as successful as medical men, but we think this very doubtful. It may further be urged that the public has not yet become accustomed to them, but as their ministrations are mainly confined to women and children, we are not disposed to attach any great weight to this plea. They have proved their intellectual fitness for passing examinations; are they equally adapted to meet the emergencies of professional life? Many will agree with Professor von Bergmann who has recently stated that he is decidedly opposed to women entering the profession. He says they possess neither the physical nor the mental attributes necessary for such arduous work. He is ungallant enough to add that so long as women are unable to beat cooks and tailors at their vocations, which women are apt to regard as their own specialities, so long will they be unable to compete successfully with medical men. He admits that women are able to do good scientific work, but he would be sorry to see them confront the exhausting strain and often the terrible despair that beset the medical practitioner's career. He winds up by saying that he has too high a regard for women to encourage them to become doctors. The women will probably not thank him for what they will probably deem misplaced chivalry. We give the distinguished surgeon's opinion for what it may be worth. As for ourselves, we do not think the experiment has been tried sufficiently long, or on a large enough scale, for a final judgment to be arrived at as to the fitness of women for the medical profession.

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Practical Notes.

THE BRONCHI.—The trachea divides into the two bronchi at the level of the upper border of the fifth dorsal vertebra. In this neighbourhood, the trachea is fixed in position so that there is no dragging on the roots of the lungs and the bronchial tubes during the various movements of the head and neck. According to Henle, there are from six to eight cartilaginous rings in the right bronchus, and from nine to twelve rings in the left, the left bronchus is consequently, as a rule, twice as long as the right. The right bronchus, according to Braune and Stahel, is wider than the left in the proportion of 100 to 78·4; this asymmetrical condition is no doubt due to the fact that the right lung is larger than the left. As the right bronchus courses towards the hilus of the right lung, it takes a much more vertical course than the left, that is, it lies more in line with the trachea. Merkel points out that the more horizontal course of the left bronchus is probably due to the fact that the heart projects to the left side of the mesial plane. On account of the straighter direction of the right bronchus and its greater width, it will be readily understood that foreign bodies such as marbles, buttons, coins, etc., which have accidentally entered the larynx and trachea, are more liable to lodge in the right bronchus than in the left. The wall of the bronchi is composed of a strong and dense fibro-elastic membrane, embedded in the substance of which are the hyaline cartilaginous rings; in the large bronchi, these are horseshoe-shaped, being deficient behind, similar to the condition found in the trachea. To the free extremities of these rings behind are attached bundles of plain muscle fibres. In the smaller bronchi, the horseshoe-shaped cartilages are replaced by small cartilaginous plates, which are also embedded in the fibro-elastic membrane. The plain muscle too is present in a relatively larger proportion than in the large bronchi. It will be observed then, that, if this plain muscle contracts the lumen of the bronchi, smaller bronchi and bronchioles will become very considerably reduced. Within the muscular layer is the submucous tissue, and internal to that is the mucous membrane.

The pneumogastric is the efferent or motor nerve to the unstriped muscle of the bronchial tubes, in animals, stimulation of the peripheral end of the cut vagus nerve causes partial occlusion of the smaller bronchial tubes, and therefore a diminished amount of air into the lung. This will in part explain the nervous side of spasmodic bronchial asthma, for the attacks of dyspnœa, which characterise bronchial asthma, are due to the spasmodic contraction of the bronchial muscle, possibly due to some abnormal stimulation of the central origin of the vagi. The chief purpose of this unstriped muscle is, in all probability, to regulate and adapt, to some extent, the quantity of air admitted to the lungs, and to each part of them, according to the supply of blood. Gairdner has pointed out that the muscular tissue also contracts upon, and gradually expels, collections of mucus, which, at times, accumulate in the bronchial tubes, and which cannot be ejected by forced expiratory efforts, owing to collapse, or other morbid conditions of the portion of lung connected with the obstructed tubes.

THE BRONCHIAL MUCOUS MEMBRANE.—The large and medium-sized bronchial tubes are lined by a columnar-ciliated epithelium which rests upon a basement-membrane. The cilia, which are prolongations of the cell protoplasm, work towards the pharynx, so that there is a constant upward movement of mucus. Between the ciliated cells are permanently differentiated cells called goblet cells, or mucus-secreting cells. They secrete fine granules of mucinogen which is extruded from the cells as mucus. External to the basement-membrane is loose tissue, containing elastic fibres and lymphoid tissue as well as racemose mucous glands, the ducts of which open on to the free surface between the columnar ciliated epithelial cells. In the bronchioles, the epithelium becomes changed from columnar and ciliated to cubical and non-ciliated, and this latter becomes continuous with the flattened epithelial cells lining the lung infundibula. It is here in the bronchioles that, as the epithelium changes its character, the plain muscle becomes more in evidence.

ACUTE BRONCHITIS.—This acute catarrhal inflammation of the mucous membrane of the large and medium-sized bronchial tubes is a particularly fatal disease to young children and old persons, because of the tendency of the inflammation to spread downwards involving the bronchioles, where the condition is known as capillary bronchitis, and usually ends in broncho-pneumonia. The disease is probably due to a micro-organism, and is particularly liable to attack those who live sedentary lives especially in ill-ventilated and overcrowded rooms. There are those, too, who seem to have a hereditary predisposition to acute attacks of bronchial catarrh, and, in whom, exposure to cold and especially north-easterly winds readily brings on an attack. It is frequently simply an extension downwards of an acute coryza, especially occurring during the late winter and early spring months. Sometimes it occurs in epidemic form.

It may be present at the onset of enteric fever and malaria, and it is a frequent complication of influenza, measles, whooping-cough, and bronchial asthma. Osler states that persons affected with spinal curvature are specially liable to the disease. The columnar-ciliated epithelial cells of the bronchial tubes become cast off; the submucous tissue becomes infiltrated with leucocytes and is generally œdematous, and the mucous glands are much swollen. There is no doubt that much of the difficulty, experienced in expelling the increased mucus from the bronchial tubes, is due to the desquamation of the ciliated cells from their lining. In the very early stages of an attack of acute bronchitis, a hot bath is beneficial, but the patient must immediately afterwards go into a warm bed, and lie between the blankets. Many persons, when they feel that they have a "cold coming on," take a Turkish bath, but this is not to be recommended, for there is great risk to the patient from exposure during the return home. After the patient is comfortably in bed, he should take a good drink of hot lemonade. A very hot linseed meal poultice, which contains some mustard, should then be applied to the front of the chest. It is best to have nothing but a piece of fine linen between the linseed and the skin, in order to give the chest the full benefit of the heat. The patient often experiences great relief from the poultice. If he is worried by a dry hacking cough, relief may be obtained by taking 10 grains of Pulvis Ipecacuanhæ Co. The air of the room must be frequently renewed; this may be done by keeping the windows open at the top, at the same time the air should be kept warm and moist by the use of the steam kettle. During what is called the dry stage of acute bronchitis, when the patient has a racking cough and a sense of rawness in the chest, the following mixture will be found to be extremely useful :—

℞.	Vini Antimonialis - - -	♏vi.
	Vini Ipecacuanhæ - - -	♏x.
	Liquoris Ammonii Acetatis - -	ʒij.
	Spiritus Ætheris Nitrosi - -	♏xv.
	Aq. Chloroformi - -	ad ℥j.

Misce. Ft. Mistura.

Two tablespoonfuls to be taken every four hours.

Following out the late Sir Andrew Clark's teaching, that it is necessary "to cause the bronchial tubes to sweat," Sir William Whitla suggests that the following may be administered after the first twenty-four hours :—

R.	Antimonii Tartarati - - -	gr. i.
	Liquoris Morphinæ Tartarati - -	ʒij.
	Vini Ipecacuanhæ - - -	ʒij.
	Aq. Camph. - - -	ad ℥vi.

Misce. Ft. Mist.

One tablespoonful to be taken every three hours.

He says, however, in the case of children, that it is unwise to prescribe opium and morphia, so for a child two years of age, the following mixture is useful :—

R.	Vini Antimonialis - - -	ʒj.
	Vini Ipecacuanhæ - - -	ʒij.
	Liquoris Ammonii Acetatis - -	ʒiv.
	Syrupi Tolui - - - -	ʒiv.
	Aq. - - - -	ad ℥ij.

Misce. Ft. Mist.

One teaspoonful to be taken every two hours.

Directly the cough becomes easier, and the mucus from the bronchial tubes more readily expectorated, the Vinum Antimoniale must be discontinued. Sometimes a stimulant is required in these circumstances. Some preparation of ammonia is indicated, and the following may be prescribed:—

℞.	Ammonii Carbonatis - -	- gr. iij.
	Spiritus Ammoniæ Aromatici -	- ♏xx.
	Aq. Chloroformi - -	ad ℥i.

Misce. Ft. Mist.

Two tablespoonfuls to be taken every four hours.

Every effort must be made to enable the patient to get rid of the mucus and mucopus, which accumulate in the bronchial tubes, and so long as there is any secretion collected there, no drugs must be administered to allay the cough. As a rule, a cough is produced reflexly by an irrritant, and so long as the irritant is there, the cough will persist. The cough, then, should not be stopped by sedative drugs, which diminish the sensibility of the bronchial mucous membrane, but expectorants administered in order to enable the patient to get rid of the *cause* of the cough.

MEASLES.—The eruption of measles, as a rule, does not appear until the fourth, or occasionally not until the fifth day after the onset of the fever, when the small red spots appear first upon the forehead and then spread to the rest of the face. The rash of measles is not always easily recognised as such, but a sign, said to be of great diagnostic value, is the early appearance of a buccal eruption. Koplik, of New York, first published his account of this eruption in 1896, and the eruption is generally known as Koplik's sign. "The unit of the eruption is a bluish-white speck upon a red ground; only a few of these may be present, or the mucous membrane may be fairly peppered with them. Often they are not detected except after careful search, for which strong sunlight is necessary; artificial light is not satisfactory. The spots are best seen on the inside of the cheeks opposite the molar teeth, and, in most cases, only there; but they may be present on almost any part of the buccal mucous membrane. Their diagnostic value is due to the fact that they are nearly always present, that they are not found in other diseases, and that they usually appear two or three days before the the skin eruption. They generally disappear at the time of full eruption" (Holt). The importance of these spots depends first upon their early appearance, and, in the second place upon their constancy in the disease. Heubner says they are present in six-sevenths of all cases; Balme says he observed them in 97·7 per cent. of 214 cases; Holt states that he had the opportunity of studying this sign in two epidemics of measles, which occurred at the New York Foundling Hospital. Careful notes were kept of the second epidemic of 187 cases. Koplik's spots were present in 169 cases, doubtful in 10, absent in 8. Holt further notes that Koplik's sign is of value in enabling the practitioner to diagnose measles from one to three days *before* it is possible to do so by the skin eruption, also in furnishing a new means of distinguishing measles from other eruptive fevers, as well as from drug rashes and those due to antitoxins.

There are numerous complications and sequelæ of measles; these, as a rule, represent an aggravation, and in some cases an extension of some of the catarrhal symptoms which normally accompany the attack of measles. Those most frequently met with are acute bronchitis, capillary bronchitis ending in broncho-pneumonia, membranous laryngitis, whooping-cough, diarrhœa, and marasmus. Sometimes ulceration of the cornea takes place, and inflammation of the middle ear is occasionally noted, which leads to perforation of the membrana tympani, and even disease of the mastoid antrum and the mastoid air cells, which may lead to lateral sinus thrombosis, meningitis, and convulsions. Goodhart and Still state that a very common result of measles is cheesy degeneration of the mediastinal glands, and a subsequent tuberculosis of the lungs, generally of the miliary type. Occasionally ulcerative stomatitis arises as a result of the catarrhal inflammation of the mouth, in which case shallow ulcers with a red base may be seen on the mucous membrane of the cheeks, lips, and gums. Extensive sloughing sometimes ensues, a condition known as cancrum oris; this usually terminates fatally.

If the rash of measles does not come out well, warm drinks and a hot bath will often hasten its development. The bowels must be kept free, and the patient confined to bed and kept there for three days after the fever subsides, and indoors in well-ventilated rooms for three weeks, unless the disease has been mild and the weather is warm.

In all cases, the mouth and nostrils should be carefully cleansed, and, during desquamation, the skin should be oiled three or four times daily with 1 in 40 carbolic oil, and warm baths given to aid the process. After all it must be remembered that the stage of convalescence from measles is perhaps the most important in the disease, for unless the child is carefully looked after and properly nursed during this period, bronchial and pulmonary complications are likely to arise which may be followed by tuberculosis. For the troublesome cough of measles the following expectorant may be prescribed :—

R.	Vini Ipecacuanhæ	ʒij.
	Spiritus Ætheris Nitrosi	♏xl.
	Glycerini	ʒiv.
	Syrupi Tolui	℥j
	Aq.	ad ℥iv.

Misce. Ft. Mist.

A teaspoonful to be taken when the cough is troublesome.

For many weeks after an attack of measles the health of the patient must be very carefully watched. Cod-liver oil and iron may be prescribed, and plenty of bracing air. The child should be well fed on good milk, eggs, and a liberal allowance of cream. During convalescence every precaution must be taken to guard against the onset of tuberculosis. It must be remembered, however, that measles is apt to take a severe form in tuberculous and rachitic children, in those subject to chronic bronchitis, and in those, too, who have recently recovered from whooping-cough, scarlatina or diphtheria.

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Preparations.

SPECIAL PREPARATIONS.

(Messrs. Parke, Davis & Co., Queen Victoria Street,
London, E.C.)

Essence of Calisaya Bark.—A distinctive feature of cinchona calisaya is that it is richer in quinine than in other alkaloids, and is consequently particularly suitable for prescription. Each fluid ounce of the Essence represents 24 grains of the highest grade of yellow Peruvian bark, blended with special flavouring and stimulating agents, thus rendering it very palatable, and enhancing its tonic value. It is supplied in bottles of 8 and 14 fluid ounces, and the dose is from **1 to 4 fluid drachms.**

Adrenalin and Eucaine Tablets.—Each of these tablets contains [illegible] gr. of Adrenalin, $\frac{1}{6}$ gr. Eucaine Lactate, and [illegible] gr. Sodium Chloride, and, when dissolved in 17 minims of cold recently boiled distilled water, makes an excellent haemostatic and analgesic agent for use in dental extractions and small operations. A tablet dissolved in 85 minims, or, say six in a fluid ounce of water, makes a solution similar in strength to that used in hospital. The tablets are issued in **tubes containing 25 each.**

Elixir Heroin and Terpin Hydrate.—This preparation contains, in each fluid ounce, $\frac{1}{6}$ gr. heroin and 8 gr. of terpin hydrate. The former is employed as an anodyne, sedative, and respiratory tonic in inflammations of the nose, throat, and bronchi, and in asthmatic and neuralgic seizures; the latter is a derivative oil of turpentine, and is used with success in chronic and sub-acute bronchitis, materially assisting expectoration. In combination, as in the elixir, they are especially useful in acute and chronic bronchitis, pneumonia, pleuritis, asthma, laryngitis, etc., as they form a pleasant and effective expectorant, exerting soothing and toning influences without the drawbacks of opium or morphine. The elixir is supplied in 4, 8, 16 and 80 fluid ounce bottles, and the dose is from **1 to 4 fluid drachms, swallowed slowly.**

ANGIER'S EMULSION.

(The Angier Chemical Co., Ltd., Snow Hill, London, E.C.)

In its unpurified state, petroleum could only be used as an external application, but, by a special process, the Angier Chemical Co. have succeeded in producing a petroleum emulsion, by which the petroleum is not only deprived of all objectionable properties, but is also rendered acceptable to the palate. Bronchial affections, especially those attended by hypersecretion of thick mucus, are particularly amenable to treatment by this emulsion. Following its administration, the expectoration becomes freer, the pulmonic circulation is assisted, cough is relieved, and general improvement secured. It is also indicated in many other respiratory affections.

The emulsion is quick to allay symptoms of irritation of the genito-urinary organs. Cystitis, urethritis, gravel and the urinary symptoms incidental to enlarged prostate, and other senile changes are quickly relieved by the antiseptic and detergent action of petroleum.

An ounce of the emulsion contains ⅓ of purified petroleum and 9 grains of the combined salts of lime and soda. The dose for adults is two teaspoonfuls.

VILLACABRAS.

(Agent: Mr. L. Verdier, 22, Gray's Inn Road, London, W.C.)

This is a natural Spanish mineral aperient water, the property of a French Company, and approved by the Academy of Medicine of Paris. It is especially useful for persons who are obliged to lead a sedentary life. The analysis of the water (as approved by the Paris Academy) is as follows:—

Sulphate of Soda - -	122·0500	grammes.
,, ,, Magnesia -	·9847	,,
,, ,, Lime - -	2·0005	,,
Chloride of Sodium - -	·9050	,,
Silica, Alumina and Iron -	·1180	,,
	126·0582	,, per litre.

It is filtered according to the Pasteur system.

THE PRACTITIONER.

MARCH, 1907.

THE DIAGNOSIS AND TREATMENT OF INFECTIVE THROAT CONDITIONS.

By MEREDITH YOUNG, M.D., D.Sc., D.P.H.,
Medical Superintendent, Stockport Corporation Hospitals.

CONDITIONS of the throat, which either are or appear to be infective, are often the cause of very considerable trouble to the general practitioner, and very frequently place his reputation at stake. Having had, for nearly fifteen years, the opportunity of examining and treating such conditions, and having frequently been placed in the unenviable position of judge of appeal in doubtful cases of this character, I believe that the result of my experience may be of assistance to other practitioners.

Simple scarlatinal sore throat is usually unmistakable, with its deep reddening of the palatal arches, uvula, and tonsils, and the not infrequent extension to the pharynx and roof of mouth. There is, I think, never any invasion of the larynx. The colour is usually a vivid red, and is, in practically every case, symmetrical. This redness lasts for a considerable time, often from 10 to 14 days, and disappears very gradually. There is almost always along with it marked pain on swallowing. From this condition alone, when at all well marked, it is possible in most cases to diagnose scarlatina. But, even in the absence of deposit, a scarlatinal throat may be confounded with simple or septic tonsillitis, measles, initial smallpox, diphtheria, secondary syphilis, and so forth. The characteristics of these may be set out in tabular form, leaving out of the question for the moment the deposit which is important enough to require special remark.

	Distribution.	Colour.	Symmetry.	Pain.	Glands.	Local Accompaniments	Pyrexia	Progress.
1 Scarlet Fever	Palatal arches, uvula, tonsils, and sometimes pharynx and roof of mouth. Buccal mucosa usually normal. Does not involve larynx.	Vivid red.	Symmetrical	Considerable	Usually unaffected in early stages: enlarged, and may suppurate later	Strawberry tongue	Marked	Disappears slowly.
2 Simple Tonsillitis.	May extend over fauces, palate, and pharynx. Larynx not involved	Deep red and almost shiny.	Generally bilateral	Considerable	Gland at angle of jaw usually enlarged and painful at an early date.	Furred tongue and foul breath.	Marked.	Disappears rapidly.
3 Septic Tonsillitis.	Rarely extends beyond base of soft palate: one or both tonsils affected	Deep red: often almost purple.	Usually, though not always, unilateral	Very severe.	Slightly enlarged at early date, but quickly subside.	Thick furring of tongue.	Marked.	Usually slow: tonsillar abscess.
4 Follicular Tonsillitis	As above	Bright red not so deep as 2 and 3	Almost always bilateral.	Fair amount.	As above	As above.	Slight.	Somewhat slow disappearance.
5 Diphtheria	Extremely variable, but usually settles on tonsils first. Larynx occasionally involved.	*Very faint reddening*	Asymmetrical	Usually *slight or absent*	Frequently enlarged, but suppuration very rare.	Typical odour: frequent croupous symptoms or nasal discharge: subsequent local paralyses.	Keeps in the region of 102 F.	Rapid disappearance (lasts a few days only).
6. Syphilis -	Involves all fauces, palate, pharynx, and larynx.	Bright red.	Symmetrical	Little or none.	Very seldom enlarged.	Ulceration: mucous patches: sulci on tongue, etc.	None.	Persistent but rapidly clears up under treatment.
7. Measles	Palate appears to be chiefly affected but only slightly so.	Red blush with tiny red papules.	Symmetrical.	Slight or none	Normal	Koplik's spots.	Marked.	Rapidly subsides.
8. Thrush -	Variable.	Normal.	Asymmetrical	None	Normal.	Patches on tongue, buccal mucosa, etc.	None.	—

The *deposit or exudate* may be classed (roughly) in a similar manner.

—	Colour.	Consistence.	Ulceration.	Persistence.
1. Scarlet Fever	Yellowish white, or dirty, and sloughing.	Very soft and easily broken up.	Distinctly present.	Disappears very slowly.
2. Septic Tonsillitis	Whitish or yellowish.	Mucoid.	Very slight.	Disappears fairly rapidly.
3. Follicular Tonsillitis.	As above.	As above.	Very rare.	As above.
4. Diphtheria -	Whitish but may be brown, or almost black owing to hæmorrhage	Tough and adherent.	Slight.	Rapid disappearance.
5. Syphilis	Dirty.	Fairly tough removal often followed by bleeding.	Almost invariable and frequently extensive: symmetrical, superficial, and often kidney-shaped.	Not of long duration.
6. Thrush - -	Dead white.	Soft and easily removable.	None.	Brief.

It is only necessary to add to the above a few further points which may help to clinch the diagnosis.

In septic tonsillitis, the onset is usually sudden, without the vomiting which ushers in scarlet fever; the pulse is rapid only with an increased temperature and keeps pace with the latter. A rash may accompany the local affection, but is always erythematous, and not punctiform, and rarely lasts more than a day.

Follicular tonsillitis, though perhaps most commonly confounded with diphtheria, is, as a matter of fact, an affection which ought to be more readily distinguished from it than anything else. The points in common are usually the low temperature, the rarity of ulceration, the fairly rapid disappearance of the local patches and the slight involvement of glands. But in four particulars the conditions clearly differ.

1. In follicular tonsillitis, the patches are mucoid and are easily removable, sometimes by mere douching with weak salt or boracic solution, practically always by swabbing with cotton wool. In diphtheria, the patches are tough and only removable, if at all, by firm and repeated swabbing.

2. In follicular tonsillitis, the patches rapidly reappear after being cleared away, in fact they may do so in the course of half-an-hour: a careful observer may actually watch the

reappearance and growth of the lacunar exudate. This is by no means the case in diphtheria.

3. The mucosa is a fairly bright red colour in follicular tonsillitis, whereas, in diphtheria, it is only faintly, if at all, reddened.

4. In follicular tonsillitis, the edges of the exudate taper off gradually, in fact there can hardly be said to be any edges at all: in diphtheria, the exudate has distinct cliff-like edges frequently curling up.

In diphtheria, it is often possible to diagnose by the facies of the patient alone; there is a singular placidity well described by Dr. H. Biss (*Lancet*, Nov. 7th, 1903, p. 1285); the onset is gradual and indefinite; there is almost always an absence of pain; prostration, pallor, and weak pulse are next door to invariable. The temperature shows an inclination to hover in the neighbourhood of 102° F. I am in the habit of resorting to alliteration in describing diphtheria to my staff at the hospital, and though exaggeration is inevitable, the quantum of truth warrants repetition: "A prostrate, pallid, pulseless, placid, painless patient, with a putrid breath and a temperature about 102°, usually means diphtheria."

As between scarlet fever and diphtheria, a high temperature, rapid pulse, delirium, and œdema of parts, over which membrane may appear, would point to scarlet fever, whilst a moderate degree of pyrexia, slowish pulse, no delirium, and a fairly normal condition of the subjacent mucosa would indicate diphtheria.

The other conditions require no further description.

Bacteriological examination has not been referred to, for, though frequently helpful, it is not, for the general practitioner at all events, of much immediate assistance.

Syphilis.—In constitutional syphilis, the throat is of a dusky red colour, and uniformly injected in the early stages: this is commonly followed by the formation of tonsillar ulcers almost circular in shape, rather deep, and with a greyish surface, the remainder of the tonsils appearing normal. If the ulceration is present on the soft palate or pharynx, and the patient is an adult, and particularly if the surface of the ulcers appears yellow and sloughy, with a thin purulent discharge, one may say, with a fair amount of certainty, that there is tertiary syphilis.

There is further a *syphilitic erythema* of the throat which should, however, be readily differentiated by the following characters from a simple catarrhal pharyngitis or tonsillitis. It is symmetrical, with fairly well-defined margins, and, owing to degenerative changes in the epithelium, which ultimately end in the formation of a mucous patch, one finds small pale bluish-white streaks or patches appearing and gradually becoming more marked, until a series of symmetrical mucous *plaques* are formed on the soft palate and fauces, or on the buccal mucosa. The coppery hue, so often spoken of in connection with syphilitic eruptions, has no application in the case of mucous membranes, though many have described it in such situations. It *may* be present, but, seen against a background of mucous membrane, it is unrecognisable.

Faucial Erysipelas has as an almost constant accompaniment, the existence of considerable œdema, a fact which, practically speaking, settles the diagnosis.

As quite distinct from any of the conditions described, one must now recognise, as a separate infectious disease, *septic sore throat due to milk.* Outbreaks of this character have been recorded at Aberdeen, Dover, Rugby, Paisley, Lincoln, Hackney, and other places. In the Dover outbreak, the local symptoms were simple inflammation, with glandular enlargement, and with vesicular eruptions on the trunk and extremities, occurring either before, or along with these local symptoms. In the Paisley outbreak (I am indebted to Dr. Robb, the Medical Officer of Health, for special information as to this) there was "an acute inflammatory condition of the throat accompanied by very severe constitutional symptoms (toxæmia), which lasted for from two to four days. It commenced with a feeling of chilliness, pains in the head, back and limbs, followed by rapid prostration. The tonsils became enlarged, and a distinct white tenacious diphtheritic-looking membrane formed on them, and extended sometimes to the uvula and soft palate, and, at other times, over the back wall of the pharynx. The temperature of the patient was high and the pulse rapid. There was no fatal result, but the patient was often left in a very weak condition. Numerous swabs from the throats of patients were examined bacteriologically, mainly because the illnesses were suggestive of diphtheria. In no case, however, was the diphtheria bacillus found, but an organism known as a diplococcus was present. Probably this organism was the cause of the disease, but we were unable to fully prove it." (*Annual Report*, 1904.)

In a further communication to the writer, Dr. Robb states that in none of the cases were there any accompanying rashes or eruptions. Diplococci were present in the throats of the patients in great numbers, and these organisms stained readily with Löffler's stain, fuchsin, and by Gram's method. The adjacent sides of the diplococci were flattened. On agar, whitish raised round colonies appeared ; growth was not very abundant and colonies tender to coalesce along the needle track. In preparations from the cultures, the diplococci were often in clumps, but sometimes were in short chains.

An outbreak at Lincoln, in 1902, showed unusual symptoms, viz., sore throat accompanied by erythema of the face, and, in two-thirds of the cases, by a roseolous papular eruption, in some cases almost urticarial ; the throat condition resulted in tenderness and swelling of the cervical glands. The difference, it is said, between this illness and scarlet fever was quite pronounced. (*Annual Report*, M. O. H. Lincoln, 1902.)

On the other hand, an outbreak at Bedford, in the same year as that named above, presented more the characters of influenza (headache, backache, pains in limbs, and temperature of about 102°) ; along with this, redness and swelling of fauces, palate and uvula with numerous patches of exudation, and, in a few cases, ulceration. The illness was accompanied or followed by considerable prostration. (*Lancet*, 1902, Vol. II., p. 1391.)

In the case of the Hackney outbreak, the Medical Officer of Health, Dr. King Warry, made a full investigation and reported to his authority, from which report the following is an extract : "Symptoms.—(*a*) In every case there was tonsillitis (not follicular). (*b*) In two cases, there was in addition superficial ulceration of the tonsil. (*c*) In every case, there was considerable swelling of the cervical lymphatic glands, more marked on the one side than the other ; in two cases, the swelling was very great. (*d*) In one case, the lymphadenitis proceeded to suppuration. (*e*) In every case, the temperature was raised, in a few cases being 105° F., and there were rigors. In all the cases, the temperature persisted after the tonsillitis had disappeared, lasting in nearly all for at least a fortnight. (*f*) The temperature assumed the remittent type, and there was, in every case, profuse sweating at nights. (*g*) There was great prostration in most cases, and considerable cachexia in all. (*h*) In one case, acute septicæmia supervened, followed by acute septic pneumonia and death. (*i*) In two cases in the same family, acute nephritis with hæmaturia came on about a week after the onset of symptoms. (*j*) In one case, a child had an attack of purpura hæmorrhagica. (*k*) In all cases, convalescence was protracted. In addition there was a marked tendency for multiple cases to occur in families, and, while no age or sex was exempt, a very large number of the sufferers were children; these features helped to direct suspicion to the milk."

Acute pneumococcal inflammation (angine érythémateuse pneumococcique) may closely simulate diphtheria, and Dr. H. Pasteur has described a typical case of this so well as to merit careful perusal. (*Lancet*, May 27th, 1905.)

Dr. Pasteur remarks:—"The local signs of pneumococcal pharyngitis are in no sense distinctive. The membranous form may closely simulate diphtheria, and a bacteriological examination alone reveal the true nature of the case. Somewhat similar appearances are also met with in some cases of streptococcal inflammation of the throat, but here the exudate is usually thinner and looser in texture, and less adherent, than in pneumococcal or diphtheritic inflammations. More reliance may be placed, in my opinion, on the early symptoms and mode of onset. In pneumococcal sore throat, the onset is usually quite sudden, often with rigor or chills, the initial symptoms are severe, and the temperature rises rapidly to 103° or higher, a grouping of symptoms, strikingly like those of pneumonia, and very rarely met with in anything like the same degree in diphtheria, or even in streptococcal pharyngitis. The disease tends to run a short course—from two to five days—but to this there are exceptions. The erythematous form of the affection is most likely to be mistaken for scarlatinal angina, and here the diagnosis, apart from bacteriological findings, must rest mainly on the absence of other signs of scarlet fever. It is worthy of note that a considerable number of the recorded cases of pneumococcal sore throat have been in adults."

Streptococcal inflammation of the fauces is perhaps only clearly distinguishable from the pneumococcal variety by bacterial diagnosis, though, from accounts of the very few cases published, the following points would indicate the latter in distinction to the former: persistence of temperature, cerebral irritation, and local sloughing.

A throat condition, which is often put down as infective, is that due to the inhalation of drain or sewer gas. In reality it is not infectious, but only appears so from the fact that several people in the same house being exposed, at or near the same time to the same effluvium, develop the same condition. The characteristics of this "drain throat" are inflammation of the tonsils, with foul tongue, intense headache, marked depression, some digestive disturbance, and a low or very slightly raised temperature.

Treatment.—As a general rule, a fairly brisk saline purge is a *sine qua non*; its effect, in simple and septic tonsillitis, is distinctly beneficial. In scarlet fever and diphtheria, I very seldom give a purgative during the first week, for, at times, they have seemed to have a malign influence on the course of the disease; the patient has often drifted from a simple attack into a septic state shortly afterwards. Whether the aperient has been responsible for this or not, I know not, but I now use a simple enema for these two classes of case, and have not observed any such bad results after its use.

As general medicines, salicylates, in my opinion, take first place for simple, septic, and follicular tonsillitis, but they are to be avoided in diphtheria, on account of the depression and possible effect on the kidneys. Constitutional treatment in syphilis is, of course, of infinitely greater importance than any local applications. In diphtheria, antitoxin, *early and in large doses*, is imperative. Diphtheria antitoxin is also frequently of the greatest use in clearing up cases of post-scarlatinal rhinorrhœa, and here a few injections of 4,000 units suffice: such rhinorrhœa is usually, if not always, associated with the presence of true diphtheria bacilli in the nose. In many throat affections, and particularly where rheumatism is an accompaniment—a somewhat frequent occurrence—I have had excellent results with salophen, or acetyl paramidol $C_{11}H_{13}NO_4$, a crystalline substance containing 50 per cent. of salicylic acid. Similar good results have followed the use of this drug in cases of influenzal sore-throat, which takes the form of acute tonsillitis, often going on to ulceration. It is usually advisable to give it in doses of ten to fifteen grains every three or four hours. Its use may be continued over a fairly long period, without the production of any stomach disturbances, such as are caused by the ordinary salicylates. When such sore throats are accompanied by much pain, salophen is particularly useful. It is also well adapted for use in the case of children owing to its tastelessness and perfect safety.

For the constitutional weakness, resulting after throat illnesses, iron, strychnine, etc. are of course required.

Local. One has always to remember, in this connection, the fact that what appears to be merely a throat affection may really be dependent on, or may eventually lead to, an affection of the nose or ear, using all these terms in their widest sense. Even if an active therapeutic advance on adjacent territory, therefore, is not necessary, still it is always imperative that scouting operations should be continuously carried out.

In the next place, one must be careful to select remedies to suit the patient's age and intelligence. In very few children, for example, can douching or irrigation, particularly of the nose, be successfully carried out, whilst gargling is a matter of such universal difficulty as to become negligible as a mode of treatment. And, finally, it must always be remembered that but little good is effected by applying remedies, unless the mucus, or other covering of the diseased area, is first of all removed. The late Sir Thomas Grainger Stewart used to tell us as students that, in treating skin diseases where crusts or scabs had formed, it was as much use putting the curative agent on the scab as on the patient's coat sleeve, and my experience is on precisely the same lines in cases of throat and nose affections.

First, then, the best means of *cleaning the area* to be treated must be considered.

Borax, boracic acid, bicarbonate of soda, common salt, sodium sulphate, salicylic acid and borax combined, *very weak* sulphurous acid, or liq. sod. chlorinat., or Sanitas (about 1 per cent.) are all useful, but personally I seldom go beyond a solution of sodium chloride (ʒii. or ʒiii. per pint of tepid water) or the same strength of sod. bicarb. or borax which, being inexpensive and safe, may be used freely. The thing to be remembered is that, for this part of the work, an antiseptic is not necessary, but only a bland wash, which is all the better perhaps if, like borax and sod. bicarb., it dissolves mucin. When the nose is blocked with crusts, I have found it best to paint with vaseline or boracic ointment, or, if any passage is left, to gently, and with a rotatory motion, pass a camel-hair brush covered with vaseline, etc., into the nostril: this causes profuse nasal secretion or sneezing with resulting loosening of the crusts.

For nasal or throat douching, the "Higginson syringe" is an abomination, and the ball-syringe is very nearly as bad. The douching should be carried out by means of a siphon-douche, and every patient should of course have a separate end-piece. For hospital wards, a dressing table on wheels with a douche-can, fixed on a raised iron bar, is best: experienced nurses know that, by the use of such an apparatus, their work, for example, in a scarlet fever ward, is very considerably lessened, whilst the gain to the patients is enormous.

Next, one may proceed to apply an antiseptic, astringent, etc. In the case of antiseptics, the range of choice is almost absurdly wide; for almost any disinfectant substance, if sufficiently diluted, may be used. My remarks will be confined to those which I have found specially useful, viz., Formamint, Sublamin, Perchloride of Mercury, Acetozone, Boracic acid, Ichthyol, Glyco-thymoline, and Adrenalin.

Three useful combinations, prepared in the form of compressed tablets, which I occasionally use, are the following:—

(1) Sod. Bicarb, grs. xii.; Ac. Carbol. Pur., gr. ijs., sod. Chlorid. grs. ii. Dissolve in a tumblerful of tepid water.

(2) Zinci Sulph., Alum. Sulph. aa 3 grs., Pot. Permang. grs. ii. Dissolve in one pint of tepid water. (This has the disadvantage of staining linen, etc.)

(3) Another useful combination is the following:—Sod. Bicarb., Sod. Borate, and Sod. Chloride, aa 5 grs.; Sod. Benzoate and Sod. Salicylate, aa 7·24 grs.; Ol. Eucalypt., 7·48 min.; Thymol, 7·48 grs.; Menthol, 7·96 grs.; Ol. Gaultheriæ, 7·96 min. One tablet dissolved in 8 oz. water.

These preparations may be used in various ways.

1. *Throat Douching or Spraying.*—This perhaps gives greater relief than any other method, and spraying is, as a rule, more grateful than douching. Preliminary cleansing (*vide supra*) is essential to success. For scarlatinal throat, simple tonsillitis, and syphilitic erythema, it will be sufficient to use either of the solutions made from the above-named compressed tablets, perchloride of mercury (or, far better, sublamin) 1 in 2,000, boracic acid 5 to 10 per cent., or glyco-thymoline 20 to 30 per cent. (here the solution gives greater relief, if it is used as hot as the patient can bear it). Formamint (which is described later) may also be used by dissolving three or four tablets in 4 oz. of warm water; this will contain formaldehyde in the strength of about 1 in 40,000. One of the great advantages of this last-named preparation is that it is not only a first-rate medicated lozenge, but can also be used like a compressed tablet for preparing douches, or for insufflation purposes (*vide infra*). The tablets should not be added until the nurse is perfectly ready to use the douche. It is particularly useful in septic conditions. Acetozone, about 10 grains per pint, is also useful; it is powerfully germicidal and non-irritating.

For septic or follicular tonsillitis, and for diphtheria, the solutions mentioned should be used in fully twice the strength, but care must be taken, in the case of the mercury perchloride solution, that it is only administered by a careful and trained nurse. Sublamin, which is stated to be mercuric ethylenediamene, is practically non-toxic and non-irritating, either to skin or mucous membranes, so that its use for this purpose may be very safely recommended. Ichthyol (from 1 to 2 drachms in 4 ozs. Aq. Dest.) is occasionally useful in irrigation; or the following: Ichthyol, grs. 50; Sod. Chlorid., grs. 25; Aq. Dest., 6 ozs.

In cases, in which rhinorrhœa persists in the shape of a thin serous discharge, I have often found that the addition of a little methylated spirit (about half-an-ounce per pint) to the lotion, used for douching, has had a most beneficial effect. Failing this, weak solution of adrenalin chloride (m. x. to xx. per pint of saline solution) may be tried.

2. *Swabbing of the throat* is necessary in septic and follicular tonsillitis, and in diphtheria, as well as in cases of foul

ulceration in syphilis, etc. For this purpose, I used Izal at one time in almost every strength possible, but abandoned it on account of the pain caused. Inasmuch as a substance, the local action of which will persist for some time, is needed, the choice is limited, and will probably not extend beyond the following :—

Ichthyol with an equal bulk of Aq. Dest., Formamint two tablets in one ounce of glycerine and water (equal parts), Glyco-thymoline pure, Tinct. Benzoin Co. and glycerine (equal parts), or the following :—

Iodi. Pot. Iodid., Ac. Carbol. pur., aa grs. iii., Glycerin ℥ss. Aq. Dest. ad ℥i.

Resorcin, a watery solution of about 10 grains to the ounce, combined with a little alkali, is also of considerable use for final swabbing.

When Formamint is used, the tablets should be crushed up in the dry state and rapidly added to the solution already made ; better results follow when one does not wait for the tablets to be dissolved ; in fact its use on these lines has been signally successful.

3. *Medicated lozenges* are, in my experience, almost invariably made far too strong for any except the most determined patient, but there is no doubt that, for children, and for those who want to carry out a part of their treatment at any rate whilst going about their business, they form a most useful adjunct to other treatment. Having tried all the B.P. lozenges, and most of the well-known proprietary antiseptic lozenges, I have become reduced to one and one only, a product of recent origin it is believed, at least in this country, viz., Formamint. The main ingredient in this lozenge is stated to be a combination of lactose and formic aldehyde containing 0·01 formic aldehyde by weight in 1 gramme of the total product : the combination is a loose one, and the addition of pepsin-hydrochloric acid causes the liberation of nascent formic aldehyde on the solution of the lozenge in the mouth. The lozenges are free from smell, and have a pleasant refreshing acid taste. It is the only lozenge that, in nearly 15 years of hospital practice, I have found children of almost any age, as well as adults, to take without having to be watched from start to finish by the nurse. It is of the greatest value in all the

throat conditions named, and should be given freely—*at least one lozenge to be sucked slowly every hour.* In the case of infants, Dr. Wyatt Wingrave has described an excellent mode of administration (*Lancet*, October 20th, 1906, p. 1068). A tablet of formamint, "crushed and carefully enclosed in a piece of butter-cloth, the ends being secured by stout thread and held, may, after moistening in water, be placed in the infant's mouth for a few minutes several times in the course of the day." Incidentally, it may be remarked that for thrush, this treatment acts like magic, especially when coupled with the administration of a few small doses of Hydrarg. c. Cret. I have many times tried this lozenge, and recommended it with success for throat irritation produced by too much smoking. In scarlet fever, its use from the start has appeared to lessen the incidence of rhinitis, and extension of inflammation to the middle ear, though naturally one makes such a statement with some reserve.

4. *Nasal Douching, etc.*—In the case of extension of the inflammatory process to the nose, preliminary cleansing, followed by insufflation or painting, is frequently required. The first part of this treatment has already been sufficiently dealt with. Suffice it to say here that any of the weak solutions mentioned may be safely used for nasal irrigation, but the greatest possible care should be taken that the irrigation is carried out on scientific lines, otherwise it will do more harm than good. The rule to be carried out, in order to prevent damage to the Eustachian tube and middle ear, is, of course, to make the patient *keep the mouth widely open and breathe quickly in and out* whilst the douching is being performed. A nurse, who says that patients cry out with earache after nasal douching, should be changed for one with more carefulness in her composition, or else the douching should be stopped at once.

Under insufflation, one may include the application of oily or viscous fluids, and, for this latter process, probably no better instrument can be used than the "Glaseptic Nebuliser" of Parke, Davis and Co. Strong applications are distinctly out of place for the delicate mucosa of the nasal cavities. For "*wet*" *insufflation*, if one may so term it, the following will be found useful in cases of extension to the nose of the conditions dealt with in this article.

i. Thymol menthol, or eucalyptol in liquid paraffin in a strength of 1 in 10.

ii. Adrenalin inhalant (adrenalin chloride one part in 1,000 parts aromatised neutral oil base containing 3 per cent. chloretone).

iii. Argyrol 25 per cent. solution once daily: carefully protect dress, etc., from stains.

iv. Hydrogen peroxide has occasionally given very satisfactory results.

v. Sublamin, 1 in 1,500 or 2,000 alcoholic solution once daily: especially useful in chronic rhinitis, nasal diphtheria, and in persistent rhinorrhœa following scarlet fever.

Of the above sublamin is to be most strongly recommended.

For *dry insufflations*, very finely powdered boracic acid, borax, "menthol snuff," iodoform in small amount, or, where greater stimulation is required, half per cent. of lysoform in boric acid (*Lancet*, October 20, 1906, p. 1068).

Boro-chloretone, 25 to 50 per cent. mixed with Calc. Phosph. etc., is also useful at times.

One substance should receive mention in an article, such as this, dealing with conditions which are frequently painful, and that is cocaine. In a strength of 20 per cent., it is most useful for painting the fauces (after preliminary cleansing) in cases in which the pain is so severe as to interfere with deglutition. In the case of the nasal cavity, a solution of from 2 to 5 per cent. strength, applied by means of cotton wool on the end of a probe, not only relieves the pain of acute rhinitis, but causes a disappearance of any turgescence of the mucosa. Similar applications may be made, using nasal bougies, each containing one-sixth grain of the alkaloid. Stovaine is a recent substitute for cocaine, and, generally speaking, may be used in half the strength of cocaine: it appears to have none of the harmful effects of cocaine on the circulatory system.

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SPHINCTERIC CONTROL OF THE MALE BLADDER, AND ITS RELATION TO PROSTATECTOMY.

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[With Plates IV. and V.]

THE exact muscular arrangement by which urine is retained in the bladder is a subject which formerly received much attention from anatomists and physiologists, and which now, owing to its increasing surgical importance, is attracting the attention of many surgeons.

From an anatomical point of view, the following muscles have to be considered:—

1. The internal sphincter vesicæ;
2. The external sphincter vesicæ;
3. The compressor urethræ muscle.

1. The internal sphincter vesicæ is composed of an ill-defined ring of involuntary muscular and elastic fibres surrounding the orifice of the bladder. According to Starling,[1] "careful dissection fails to show any thickening of muscle round the commencement of the urethra sufficient to constitute a sphincter. But the first part of the urethra is surrounded by a muscular coat, consisting of an inner longitudinal layer of fibres, and an outer thicker layer continuous with the inner and middle layers of the bladder." According to Reyfisch,[2] this internal sphincter may be inhibited or set in contraction by voluntary impulses.

2. The external sphincter of Henle is composed of a few transverse fibres on the ventral surface of the prostate gland. This in man is the commencement of a sheet of striped muscle which surrounds the urethra to within an inch or so of the external meatus.

3. The compressor urethræ muscle. This is the name applied to a powerful muscular apparatus, of which the inner surface consists of longitudinal and circular unstriped muscles; the outer layer of striped muscle fibres, which are continuous above with the external sphincter of Henle, and surround the

membranous urethra circularly and obliquely. This striped muscle is usually called the compressor urethræ muscle.

These muscles are, no doubt, from their position, all concerned in retaining urine in the bladder. Which is the important one to avoid injuring in the surgery of this region is a question which has provoked a good deal of controversy, different observers getting different results according to the methods employed. Now that a large number of prostatectomies have been performed, an opportunity has been afforded in man of deciding the question. The result has been that it has generally been accepted that the important muscle is the compressor urethræ muscle, and that, if incontinence is to be certainly avoided, the membranous urethra should be carefully left uninjured. This question is fully considered by E. Wood Raggles, in the *Annals of Surgery* for April, 1905, in which the following quotation from a paper of Freyer's is given: "It would thus appear that, after complete enucleation of the prostate, the prostatic urethra, deprived of its normal support, widens out in funnel fashion, and practically becomes part of the bladder cavity. It further demonstrates that the true sphincter of the urethra (or bladder) is situated in the membranous portion of the urethra."

I myself, and I suppose the majority of surgeons interested in this region, felt quite satisfied that the important sphincter muscle was that surrounding the membranous urethra until the following surprising result was obtained in one of my own prostatectomy cases.

A perineal prostatectomy was done, on a man, æt. 75, on March 8, 1905. Young's conservative operation was attempted. An incision was made in the membranous urethra, and the prostatic tractor inserted into the bladder. The prostate, however, did not enucleate nicely, and had to be removed in pieces, the prostatic urethra being largely destroyed. The mucous membrane of the bladder, however, was not injured. A double tube was inserted into the bladder, and continuous irrigation kept up for five days. The tubes were then removed, and I expected the patient to be constantly wet until the fistula closed. To my surprise this did not occur; the patient asked for the bed-pan the same evening, and passed 10 ounces of urine through the fistula. This control continued until the fistula healed, and the urine came per urethram. In this case, the membranous urethra was open in its upper part; the prostatic urethra was largely destroyed; this control, which was almost perfect, must, therefore, have taken place by means of the internal sphincter. This observation, although only an isolated one, still made me seriously consider the question anew, and I resolved to try to find out, experimentally on animals, whether the membranous urethra was so important after all.

In the dog, the prostate is very like that of man, in fact a mesial section through the urethra prostate and bladder of a dog is extraordinarily like that of a child (Fig. 1). The only apparent difference, microscopically, being the increased development of the glandular structure in the adult dog. In the commoner forms of monkey, the prostate is a small bilateral structure lying mostly on the posterior aspect of the urethra and not distributed so evenly all round the urethra as in the dog and man. I have not yet had an opportunity of dissecting this region in the anthropoid apes.

Dogs were used for the observations, and only the immediate results were considered. It is obvious that here, as in other parts of the body, the muscles left have considerable powers of adapting their function to take the place of those removed. This is one of the reasons that the ultimate perfect control, after prostatectomy in man, does not necessarily mean that the important sphincter muscle has been left uninjured.

In the dog, the prostate can be readily exposed below the bladder by a vertical incision through the lower part of the abdominal wall at the side of the penis. The insertion of the rectus muscle of that side being detached as far out as is necessary to admit the finger to the pelvis. The urethra below the prostate can then be separated from the rectum, and the prostate drawn up into the wound. The ease with which this can be done appears to depend on the age of the dog; in some the prostate is on a level with, or even slightly above, the pubis, in others it lies deep in the pelvis. From a considerable number I have examined, it would appear that the older the dog the higher is the position of the prostate with regard to the pelvis. It appears to me also that, if senile hypertrophy takes place, great enlargement might occur without producing any symptoms, because of the freedom of the prostate to expand without compressing the urethra, or encroaching on the bladder. The question of hypertrophy of the prostate in the lower animals can only be incidentally referred to here. On a future occasion, I hope to deal with it more fully. Fig. 2 is taken from a dog, known to be 11 years old, and illustrates the anatomy of the part; it shows the largest prostate I have met with in the dog.

Experiment 1.—The prostate was exposed by an incision as described above. The urethra was divided transversely a little below the apex of the prostate, with the object of stitching it to the anterior abdominal wall. To aid in this, and to render it functionless, this piece of urethra was slit up into the prostatic structure, and stitched to the edges of the wound, which was then closed. The following morning, the dog made efforts at micturition, but was unable to pass water. A catheter was passed through the prostate and the urine drawn off. After this the dog passed water apparently quite normally, so far as control was concerned, the only difference noticeable was a slight difficulty in cutting off the flow. So that, when only passing a small quantity of urine at a time, after the custom of the dog, a slight dribble took place for a few seconds. This, I think, could to a large extent be accounted for by the fact that the prostate had retracted into the depths of the wound.

Experiment 2.—In order to confirm the first experiment, the prostate and membranous urethra were similarly exposed in another dog. The membranous urethra was quite isolated. An incision was made across the posterior surface of the prostate and into the prostatic tissue, so as to make sure that all the nerves and vessels passing down to the membranous urethra were divided. The muscle fibres surrounding the urethra were then divided, leaving practically only the mucous membrane intact, in fact it appeared quite likely that the urethra might necrose.

The result of this experiment completely confirmed the first, for the dog had perfect control, and micturition was perfectly normal. It now remained to find out whether this control lay in the prostate or above it.

Experiment 3.—In a third dog, the membranous urethra was cut across as before, and the prostate removed in slices

PLATE IV.

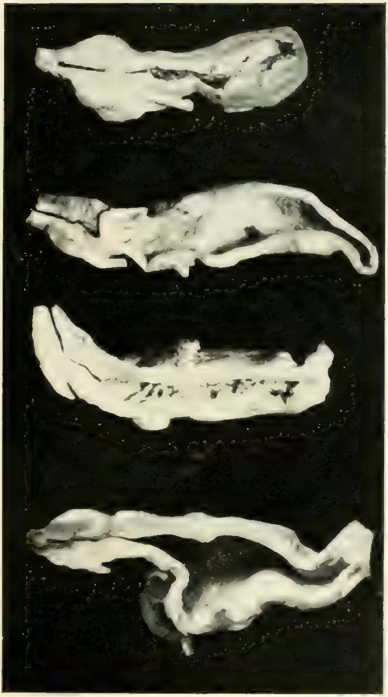

Fig. 1.—*Photographs to show the similarity between the Bladder, Prostate, and Urethra in dog and man. The first three are actual size, the fourth is reduced to make the comparison easier. The first two specimens are from dogs. The third and fourth are human, æt. 3 and 14 respectively.*

PLATE V.

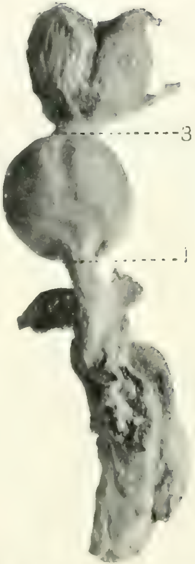

Fig. 2.—*Photograph of the Bladder, Prostate, and Urethra from a dog over 11 years old. At the top the bladder is seen cut open and contracted, below this the Prostate, which is very large, this makes the tone of the internal sphincter muscle appear unusually well marked.* 1. *Indicates the site of the "short circuit" of the Urethra in Experiment* 1. 3. *Indicates the "short circuit" in Experiment* 3.

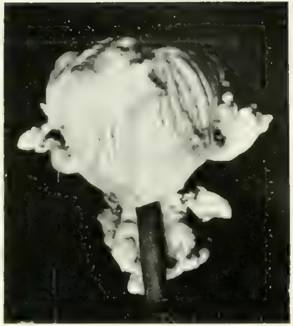

Fig. 3.—*Photograph actual size of the Bladder removed post mortem from Experiment* 3. *The Bladder, which was found to contain about two ounces of urine, was emptied by the manipulations necessary to remove it owing to the relaxation of the sphincter after death. A tube has been placed through the internal sphincter to show the amount of prostate and urethra that had been removed anteriorly.*

until the entire structure was taken away, with the exception of a small piece on the posterior aspect, which was left to give a fastening to a rubber drainage tube put in to convey the urine to the surface. I was under the impression that, anteriorly, I had injured the bladder sphincter which was relaxed and open. This relaxation possibly was accounted for by the depth of the narcosis, as by mistake a larger dose of morphia had been given hypodermically, previous to the administration of the anæsthetic, than had been intended.

I had already ascertained that the entire prostate could be sliced away with a moderately distended bladder without the urine escaping. This in itself did not seem conclusive, as the manipulation and stimulus of cutting possibly caused a spasm of the internal sphincter. The result of Experiment 3 was almost exactly the same as that of Experiment 1. There was no incontinence. When the dog was allowed to run about with the bladder distended by the urine of 6 or 8 hours, there was apparently some difficulty in cutting off the flow, more dribbling took place between the acts of micturition than in the first experiment. This difficulty of stopping only lasted a few seconds, and seemed to occur when the dog passed water with a full bladder, afterwards, when taken out for a run, micturition seemed perfectly normal.

The dog was chloroformed 10 days after the operation, and the bladder, postmortem, contained about 2 ounces of urine (Fig. 3).

The above experiments demonstrate clearly that, in the dog, the important muscle, which retains the urine in the bladder, is the internal sphincter vesicæ.

Von Zeissel[3] has shown, in curarised animals, that the internal sphincter hinders water injected into the urethra from flowing into the bladder, and that stimulation of the nervi erigentes relaxes this sphincter, and allows the flow to take place.

Finger,[1] referring to these experiments of Von Zeissel, says that "the anatomical conditions in beast and man are quite different, in beasts the mechanism of the closing of the bladder is according to its anatomical arrangements purely involuntary, in man, on the other hand, it is capable of being decidedly influenced through the will." This statement does not appear to be correct for all animals. I have already pointed out the anatomical similarity between dog and man. In the dog at least, among the animals, micturition is not "purely involuntary." Many dogs can voluntarily retain their urine for 12 or more hours. This voluntary control is, of course, developed in house dogs, but I have also seen it in sporting dogs that have had no previous training in this respect. Of course that which is true in the case of the dog must also, if possible, be shown to be so in man.

Leedham-Green,[5] by filling the living human bladder with an emulsion of bismuth, and then taking a radiograph of the pelvis, demonstrates that the full bladder is spherical, that is, that the internal vesical sphincter remains closed, and not, as held by Finger, pear-shaped (Fig. 4), owing to the pressure of the distending fluid causing the internal vesical sphincter to relax, and allowing the prostatic urethra to distend down to the compressor urethræ muscle. The views of Finger, which are so well known, and so generally accepted, because they explain many phenomena which occur in pathological conditions of the urethra and bladder, I need not deal with

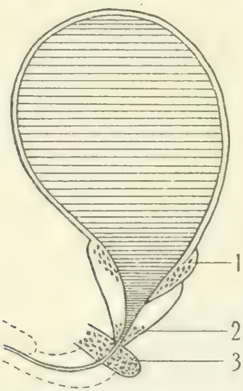

Fig. 4.—*Shape of male bladder, according to Finger, when fully distended.*[6] 1. *Internal sphincter vesicæ;* 2. *External sphincter vesicæ;* 3. *Compressor urethræ muscle.*

here, as they have recently been so fully gone into by Leedham-Green[6] in his recent paper in the *British Medical Journal*.

Oppenheim and Löw,[7] at the clinic of Professor Finger, have carried out similar experiments to Leedham-Green's on the living bladders in apes, having ascertained that they were analogous to those in man. They obtain results supporting Finger's theories.

This ingenious method of investigating the shape of the living bladder is open to the criticism that a certain amount of irritation is produced by the injection of the opaque fluid, whether a catheter is used or not. This may cause a slight spasm of the sphincter muscle, and obscure the result, particularly in the case of one of Leedham-Green's radiographs, which shows a metal bougie *in situ* with the bladder distended.

Again, in the case of the apes, the fact that a certain amount of anæsthesia was necessary possibly accounts for the fact that the internal sphincter is relaxed in the distended bladders.

Conclusions.—In order to prove absolutely that in man the internal sphincter vesicæ is the important muscle, it will be necessary to obtain observations on all cases of surgical interference with this region, particularly in young patients that have not reached the complication of an enlarged prostate, for, in such cases, the action of the internal sphincter must be considerably interfered with, particularly in those in which there is much intravesical enlargement. This interference possibly accounts for the symptom, frequently seen in cases that have not reached complete obstruction to micturition, namely, inability to retain the urine if a sudden desire to pass water takes place. When this condition arises, very likely the compressor urethræ muscle develops increased activity to counteract the symptom.

After prostatectomy, the control of micturition is usually somewhat abnormal at first, but quickly becomes apparently quite normal. This is probably owing to the compensatory powers of the compressor urethræ muscle. Whether the internal sphincter acts after supra-pubic prostatectomy is a point which no doubt will soon be ascertained by autopsies on patients some years after the operation. But it seems quite incorrect to argue, from operations on patients with enlarged prostates, that the chief sphincter of the bladder normally lies in the membranous urethra.

The foregoing experiments were carried out in the School of Physiology in Trinity College, Dublin. I have to express my best thanks to Professor Thompson, King's Professor of the Institutes of Medicine, for placing the resources of the department at my disposal, and for many courtesies extended.

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[4] *Die Blennorrhoe Sexualorgane*, 1905.

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SOME REMARKS ON HÆMOPTYSIS AND ITS TREATMENT.[1]

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In offering some remarks on hæmoptysis, I do not propose to go into the matter exhaustively. I merely desire to speak a little about the diagnosis of the more ordinary forms of true and spurious hæmoptysis, and, at rather less extended length, on the treatment of pulmonary hæmorrhage from phthisis.

The term hæmoptysis is properly and classically confined to blood-spitting, when the blood spat comes from the respiratory organs. Clinically however, we are frequently confronted by patients, in whom the blood-spitting, of which they complain, does not come from the lungs, bronchi, or trachea. Such cases would be defined as spurious hæmoptysis, but, from the practical point of view, it is more convenient to consider all such cases together.

Blood-spitting is almost indissolubly associated, in the public mind, with phthisis, and hence its occurrence, even in the mildest form, is generally sufficient to make the patient hasten at once to a doctor fully prepared to hear that he has the dread disease, consumption. It behoves us, however, not to clinch this diagnosis lightly. Let me mention a few of the different kinds of cases of hæmoptysis, real or so-called, with which I have been confronted in the course of an ordinary experience.

One kind of case occurs not infrequently in anæmic and rather neurotic young women. They are brought to us by their mothers complaining that they wake up every morning with a taste of blood in their mouth, or with their mouth full of blood, or that their pillows are stained every morning with blood.

On investigation, we find that there are no abnormal physical signs in the lungs, that the patient has usually the ordinary symptoms and appearance of anæmia, that the gums are pale and inclined to recede from the teeth, that the teeth are carious and ill kept, and that there is frequently a certain amount of pyorrhea alveolaris. The blood-spitting turns out to be saliva tinged pink by oozing from the gums.

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[1] A lecture delivered before the South Midland Branch of the British Medical Association.

Such cases are easily cured by astringent and antiseptic mouth washes, by better care of the teeth, and by the ordinary treatment for anæmia. Other cases of unimportant blood-spitting are associated with granular pharyngitis, and perhaps a certain amount of laryngitis and tracheitis, there is much hawking and perhaps a violent spasmodic cough, and occasional little pellets are expectorated streaked with blood on the outside. Another form of spurious hæmoptysis, with which I have met, has come from slight nasal hæmorrhage running backward into the pharynx at night and then being expectorated by the mouth.

Hæmoptysis from cardiac disease is rather more important since it is frequently attributed to pulmonary tuberculosis.

Not long ago I had an elderly woman sent to the hospital for open-air treatment, as she had had hæmoptysis and was supposed to have phthisis. There were however no tubercle bacilli in her sputum, no definite physical signs in her lungs, and the cardiac murmurs characteristic of mitral stenosis. Her subsequent history showed that she had not got phthisis, but that her hæmoptysis was due to her cardiac condition.

Owing to the increase of backward pressure in the lungs from mitral stenosis, this is the form of valvular disease in which hæmoptysis is most likely to occur, but it is also not uncommon in mitral regurgitations with a failing heart.

The hæmoptysis in these cases is not generally large in amount, and occurs very often in the form of small dark, purple, airless, or almost airless, lumps.

The other form of hæmoptysis, which occurs in cardiac diseases, is, of course, from infarction of the lung, from a pulmonary embolism. This is associated with definite physical signs, very often in the axillary region, and may be accompanied by a considerable amount of hæmoptysis.

Another form of hæmoptysis, not very common but of importance from a diagnostic point of view, occurs in elderly people from atheroma of the arteries and vascular degeneration.

Last year, I saw a very interesting example of this in an elderly lady. She was extremely emaciated, had a violent spasmodic cough, and had four or five times coughed up about an eggcupful of blood. She was supposed to have senile tuberculosis. On examination, however, she had a hypertrophied heart, and thickened, and probably calcareous, peripheral blood vessels; from the examination of the urine the kidneys did not appear diseased. The lungs were emphysematous, with numerous dry crackling crepitations chiefly at the base, and there was a strong family history of gout. The pulse was curiously enough not high tension. A specimen of the sputum could not then be obtained. I thought that the case was one of a gouty spasmodic bronchitis with vascular degeneration, and ventured to prophesy that hæmorrhage might very likely occur elsewhere, for example, in the brain. A little more than a fortnight afterwards she died from sudden cerebral hæmorrhage.

Hæmoptysis from more gross vascular disease occurs, of course, in aneurysms of the aorta, either in the form of a scanty leaking of blood into the bronchus, or a sudden gush immediately fatal.

Another form of hæmoptysis, which may be due to vascular changes, or to blood changes, or to both vascular and blood changes, occurs occasionally in the later stages of chronic interstitial nephritis. It is generally associated with hæmorrhage from other regions as well, and, so far as my experience goes, is of very fatal import.

An example of this I saw, two years ago, in a publican. For several weeks, he had had copious hæmorrhage from the nose, and, when I saw him, had been coughing up for two days, every few minutes, mouthfuls of bright red frothy blood. For the time being, he recovered by the aid of calcium chloride and other remedies, but died in a few months from a recurrence of the same condition. No signs of tubercle could at any time be discovered in the lung.

Of the blood-spitting, which occurs in purpura, pernicious anæmia, and other profound blood diseases, I have seen no example, although I have seen such cases in which there has been hæmorrhage from the nose, the stomach, the bowels, and the uterus. Hæmorrhage from the lungs would seem to be very rare in these conditions.

The above-mentioned cases of blood-spitting are not so much due to lung disease itself as to alterations in the blood, the blood-vessels, or the blood pressure in the lungs due to disease elsewhere.

Blood-spitting due to actual disease of the lung itself occurs in lobar pneumonia, emphysema, carcinoma of the lung, streptothrix infections of the lungs, and, last but not least, in pulmonary tuberculosis.

The nature of the expectoration in an ordinary case of lobar pneumonia is so familiar to us all that I need say no more about it. In broken-down alcoholic subjects, however, the expectoration may have a very different character, being no longer viscid but liquid, and of a dark prune juice colour. As these cases are often associated with a comparatively low temperature, and none of the characteristic symptoms of the disease, they may lead to a mistaken diagnosis.

Emphysema of the lungs is associated with atheroma of the pulmonary artery, and it is perhaps surprising that blood-spitting does not occur more often under such conditions. I do not think I have ever seen a case unless the lady, I mentioned above, who died of cerebral hæmorrhage, may be considered one. The lungs were emphysematous, but she had, in addition, degeneration of the systemic blood-vessels.

Carcinoma of the lungs, which is nearly always secondary to disease elsewhere, is frequently associated with very moderate and slight hæmoptysis, the blood being intimately associated with the sputum in dark purplish masses, the so-called gooseberry-jelly expectoration.

I saw an example of this not long ago in an elderly woman, who was sent up to the hospital, supposed to be suffering from bronchitis. She had had, however, one of her mammary glands removed several years before, and, shortly after admission, began to cough up this characteristic sputum containing cancer cells. The post-mortem examination showed the lungs full of secondary carcinomatous growths.

Streptothrix infections of the lungs, such as actinomycosis, may be associated with hæmoptysis, but hæmoptysis in these cases is, I think, much more uncommon than in phthisis. This is somewhat surprising as there are great degenerative changes in the lungs, and the blood-vessels do not tend to become obliterated as in phthisis. The sputum is, however, often of an anchovy-sauce colour, presumably due to very small traces of blood intimately mixed with it.

In pulmonary tuberculosis, hæmoptysis may occur early in the disease, or late, or both early and late, and throughout the disease, or again not at all.

When the disease is early, or comparatively early, hæmoptysis may occur, so far as my experience goes, in three forms.

The commonest is, I think, the expectoration of small masses of sputum from the alveoli, deeply stained a purplish-red from the intimate admixture of small traces of blood. Such hæmoptysis is very much like that which occurs in cardiac disease, or cancer of the lung.

Less common, but still fairly common, is the expectoration of bright red, frothy blood in small quantities, perhaps about a teaspoonful at a time, and often mixed with the sputum.

An extremely rare form occurs when the blood slowly leaks into the bronchi. In such cases, a blood cast of the bronchi may be expectorated, closely resembling the casts coughed up in fibrinous bronchitis.

I have seen one example of this in the case of a clerk at the Northampton General Hospital. He came to me complaining of blood-spitting. No abnormal physical signs could be detected in the lungs, but when at last I obtained a specimen of the hæmoptysis, I was greatly surprised to find it looking almost like a mass of convoluted worms stained a dark red. On floating this mass out in a shallow glass dish, and gently separating its component parts with a needle, it was found to form an almost complete cast of a bronchial tree with its larger ramifications. The case was associated with none of the suffocative spasms characteristic of fibrinous bronchitis, but, at the same time, no tubercle bacilli could be found. The diagnosis remained in doubt but with a strong bias towards pulmonary tuberculosis. A second cast of the same nature, but less perfect, was expectorated, and then ordinary hæmorrhagic sputum was coughed up containing tubercle bacilli, and early physical signs developed at both apices. Since that time, three years ago, the patient has had several small attacks of ordinary hæmoptysis.

The hæmoptysis, which occurs in the later stages of pulmonary tuberculosis, is, of course, familiar to us all. Bright red frothy blood is brought up with every cough; not very much at a time is usually expectorated, but, in the course of 24 hours, as much as half a pint or a pint may have been coughed up. It is only very occasionally that the hæmoptysis is so profuse as to be immediately fatal, and I think I can only remember seeing one such case.

The pathological cause of the hæmorrhage, in these late cases, is pretty obvious; it is due, in the greater number of cases, to the rupture of a pulmonary aneurysm, and less frequently to the direct softening and ulceration of the wall of a blood-vessel.

In earlier cases, the cause is not quite so obvious. Probably, as a rule, it is due to an inflammatory dilatation and rupture of minute blood-vessels in the bronchioles and alveoli, and is of the same nature as the hæmorrhage that occurs in pneumonia. It is difficult, however, to always satisfactorily explain the enormous number of hæmorrhages which occur in some cases of phthisis. I have one case, from time to time, under treatment in which there is no fever, and in which the physical signs have always been for the past two years of the obscurest character, merely consisting of a few very doubtful crepitations on inspiration at the left apex posteriorly, and yet there have been quite seven or eight attacks of hæmoptysis: another much more advanced case, but without physical signs of a cavity, in which there have been no less than seventy attacks of hæmoptysis.

These cases may be due to fresh foci of disease with congestive hæmorrhage, but another explanation has been advanced for some of them, and that is that there may be a small concealed cavity with a pulmonary aneurysm, or ulcerated blood-vessel in its walls, and that tubercularisation of the clot may occur. The aneurysm of the blood-vessel ruptures, hæmoptysis occurs, a clot is found, and the bleeding ceases; then tubercle bacilli attack the clot, soften it, and break it down, a fresh hæmorrhage occurs and sweeps away the tubercle bacilli, and another clot is formed, and so on again and again.

As regards the differential diagnosis of the cause of a given case of hæmoptysis, I wish to say one word only.

The probability is, as a rule, greatly in favour of the cause being pulmonary tuberculosis. If, however, no physical signs, characteristic of the disease, can be found in the lungs, and no tubercle bacilli in the sputum, a careful search ought to be made for the other diseases which may cause hæmoptysis. If, however, no cause can be found, then the probability is that pulmonary tuberculosis is nevertheless the "fons et origo mali."

A discussion of the efficacy of treatment in any disease is always apt to be vitiated by a great fallacy. The human body has curative powers of its own, and diseases thus tend to self-limitation. It is therefore often entirely fallacious to say that a remedy has been effectual in any given disease, because the disease improves after the remedy has been given. Hæmoptysis pre-eminently tends to a natural cure, at any rate for the time being, and it is this fallacy which, I suppose, has given rise to the extraordinary string of remedies which one finds advocated for the treatment of hæmoptysis in almost any text-book of medicine or therapeutics, remedies which, in my opinion, can either do no good or are even probably actively harmful. Take, for example, what may be called local astringents, such as perchloride of iron, alum, acetate of lead, or tannic, and gallic acid. Locally, in the stomach and intestines, they may conceivably have a powerful action, but it is very difficult to understand how they are to act in hæmorrhage from the lungs, unless indeed they are swallowed the wrong way. They are not only probably entirely useless, but indeed indirectly actually harmful, because they tend to cause constipation, which is most undesirable. Take, again, the vascular constringents, such as ergot, suprarenal extract, and digitalis. Now, both physiologically and pathologically, it is very difficult to see how such remedies can do anything but harm. Physiologically, it has been proved that the pulmonary blood-vessels are innervated, but only to an extent apparently sufficient to maintain a certain amount of tone in the vessels. Adrenalin, for example, while it powerfully constricts the peripheral blood-vessels, causes hardly an appreciable effect on the vessels of the lungs. If this is the case, then these vascular constringents raise the blood pressure in the lungs without any compensating constriction of the vessels, a most undesirable and dangerous result.

But, even supposing the vessels of the lungs were capable of contracting under the influence of these drugs, it is very unlikely that any good could be done by such contraction. The greater number of cases of hæmorrhage of any large amount are due to the rupture of a pulmonary aneurysm, or to the ulceration of a vessel of considerable size. In either case, the vascular constriction would not be sufficient to obliterate the vessel leading to the diseased area, at the same time the blood pressure is raised, and hæmorrhage would therefore probably be rather expedited than diminished. Despite, then, the reputation which these drugs have acquired in the treatment of hæmoptysis, I believe that they should be discarded, as in reality almost certainly harmful.

If we cannot influence the hæmorrhage by applying topical astringents, or by causing contraction of the vessels, what are the indications for the treatment which we can meet?

In hæmoptysis we want to give as much rest as possible to the lungs and the heart, we want to lower the blood-pressure, and we want, if possible, to increase the coagulability of the blood. How may these indications be fulfilled?

Rest to the lungs and the heart can only be given by general rest to the body. This is obtained by complete rest in bed, and by not allowing the patient to get up for any purpose, unless he happens to be one of those patients who cannot pass a motion in bed without great agitation and difficulty. In such a case it may be best to allow him to get up quietly and slowly for the purpose. Another measure which will soothe the patient, and give him bodily and mental rest, quieting the cough and relieving mental apprehensions, is morphia, given either by the mouth or under the skin.

In order to lower the blood pressure we may combine various measures.

(*a*) *Posture* will alter the effects of gravity, and prevent passive congestion of the lung. The patient should not lie flat, but be well propped up with pillows, or a bed-rest.

(*b*) *Diet*, a restricted diet, somewhat after the method of Tufnel's diet for aneurysm, will help. The diet should be cool, given in small quantities at a time, and with a diminution of liquids, and a small quantity of easily digestible solids, such as minced meat, or minced chicken in the form of sandwiches. Thirst may be combated by allowing small pieces of ice to be sucked.

(c) *Derivative medicines*, which will carry away fluid from the blood and dilate the abdominal blood-vessels. The most convenient for this purpose are mild saline purgatives. I say mild ones because violent or frequent purgation is not desirable, owing to the disturbance which it causes to the patient.

A combination of magnesium and sodium sulphate, such as occurs in the ordinary hospital house mixture, is often sufficient, or if the patient is constipated by the morphia, which is so often necessary, a 5-gr. colocynth and hyoscyamus pill in addition will generally be enough.

(d) *Vaso-Dilators.*—If we cannot stop bleeding from the lungs by vaso-constrictors, we can certainly influence the blood-pressure in the lungs by vaso-dilators, either immediately and evanescently by inhalations of nitrite of amyl, or more gradually and continuously by nitroglycerine, or erythrol tetranitrate. The expense of erythrol tetranitrate is rather prohibitive, in most cases, and I think that the best plan is to give two tabellæ trinitrini in the 24 hours after Whitla's manner, that is to say, breaking them up into small pieces, and letting the patient take a piece every hour.

The third indication to increase the coagulability of the blood may be accomplished by giving 15- to 20-grain doses of calcium chloride, either three times a day, or more frequently such as four or six hourly. This, however, according to Professor Wright, must not be given for too long, not for more than three or four days at a time, otherwise the effect will be the reverse, and the blood will become less coagulable.

These, I believe, are the chief measures which are likely to do good in a case of hæmoptysis, but there are one or two minor measures, about which I should like to say a word.

The application of ice to the chest is, I think, useless, unless indeed it is applied over the heart in order to quiet its action. It is difficult to form any postulate, by which the action of ice on the skin can improve bleeding from somewhere in the substance of the lung. I should not wish to be too dogmatic about this, but I am not accustomed to use it myself.

The inhalation of turpentine is possibly beneficial. According to Lauder Brunton, turpentine, diluted with vapour, is a vaso-constrictor, and may, therefore, act in this way on bleeding areas in the lungs, and turpentine is also said to depress the action of the heart. At any rate, I am accustomed to let my patients inhale it from time to time sprinkled on a piece of lint. If no other benefit is gained, having something to do like this quiets the patient's nerves and reassures him.

Then one more remark about posture. If the hæmorrhage is severe, and it is pretty certain that it comes from one lung rather than the other, it is best, while the hæmorrhage is going on, to let the patient lie on that side in order to prevent the other lung getting flooded with blood.

To recapitulate the treatment of a case of hæmoptysis, I should put the patient at once to bed in a cool, airy room in a propped-up position. I should reassure him, a very important point, give him an injection of morphia, and let him have some turpentine to inhale. He should be put on a calcium-chloride mixture, and have his bowels kept well open by a saline purgative. He should have a cool and partly dry diet, and ice to suck to relieve thirst. In most cases, the hæmoptysis will soon stop, but, if it is persistent or recurrent, I should add to my armamentarium the use of nitro-glycerine.

This is, I believe, the rational way of treating a case of hæmoptysis from pulmonary tuberculosis, and, in my opinion, we may discard, as not only useless, but harmful, a great number of drugs, although they have come down to us with the weight of very distinguished names behind them.

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A NOTE ON CÆSAREAN SECTION, BASED ON AN EXPERIENCE OF SIXTEEN CASES.

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This note is based on an experience of 16 cases of Cæsarean section operated upon during the last 12 years. Of these, the indication for interference in 13 was deformity of the pelvis: in the remaining 3, operation was required on account of obstruction by tumours.

The 13 operations for pelvic deformity were performed upon 9 women, six of whom underwent section once, 2 twice and 1 three times.

The varieties of pelvic deformity were as follows:—

In 1 patient, flattening of the pelvis, without signs of rickets.

In 3 patients, rachitic flat pelvis, with general contraction.

In 2 patients, general contraction of the pelvis, without signs of rickets.

In 2 patients, marked deformity of pelvis due to hip-joint disease in childhood.

In 1 case, pelvic deformity in an achondroplasiac dwarf.

In the 3 cases, in which tumours formed the obstacle to delivery, obstruction was offered by advanced carcinoma of the cervix uteri in one; by a large intra-ligamentous fibroid in another; and by a dermoid cyst of the left ovary in a third.

The cases are as follow:—

Case I.—Mrs. E., aged 33: married 15 years: pregnant for the tenth time. At the fifth month of pregnancy, a "cauliflower" carcinoma of the cervix uteri had been recognised, and from that time, until August 1894, when the patient came under my care, she had been treated in various ways, once by curetting, several times by applications of perchloride of iron and chromic acid. When I saw her, the vagina was occupied by a huge mass of growth with a foul sloughy surface. The entire vaginal cervix was involved, and the os was nowhere to be found. It was decided to perform Cæsarean section with Porro's modification, if complete hysterectomy was not possible.

August 12, 1894. *Operation.*—Eventration of uterus, the abdominal wound being temporarily closed with forceps. Cæsarean section : delivery of healthy living child. Placenta attached to posterior wall. Porro's operation with extra-peritoneal treatment of the stump. The latter separated on the 13th day, after which rapid recovery took place. The child (female) thrived and did well. The mother survived for nine months.

Case II.—Mrs. O., aged 39 : married 1½ years : was brought to see me, on April 4, 1901, by Dr. Wynne Griffith, of Pwllheli, Pregnancy had almost reached full term, and the pregnant uterus was forced over to the left side of the abdomen by a large intra-ligamentous fibroid of the uterus. The immobile tumour extended from a point 1½ ins. above the umbilicus to the pelvic brim, the right half of which it roofed over. The pelvis was flattened, the diagonal conjugate measuring 2½ ins.

Delivery of a full term fœtus, alive or dead, by the vaginal route being impossible, the patient was admitted to the Hospital for Women to undergo Cæsarean section. During the night, intermittent abdominal pains were complained of, and next day some dilatation of the cervix was noted, so operation was performed forthwith.

When the abdomen was opened, neither uterus nor tumour would budge an inch, so the incision into the former had, perforce, to be made close to the line of junction. It struck the placental edge as had been expected, but bleeding was slight, and there was no difficulty in delivering the fœtus from the fourth breech position. The uterus contracted well, and hæmorrhage was trifling. After further exploration, it was deemed advisable to rest content with the conservative operation, as the patient's condition did not justify the removal of the myomatous uterus. The uterine wound was closed by two layers of catgut sutures, and the abdomen closed. Recovery was uninterrupted, and the patient left the hospital with her healthy infant four weeks later.

Case III.—Mrs. J. J., aged 27. I gravida. Labour began on April 3, 1901. It went on fitfully and with but little progress until the early morning of the 6th, when the district midwife summoned assistance, and Dr. Inman, the obstetric assistant, saw the case. He at once recognised its gravity, and sent the patient into hospital. Soon after admission the membranes ruptured.

The patient was a feeble anæmic person, with a cachectic appearance due to syphilis. Her stature was stunted, and hip-joint disease in childhood had led to shortening of the left leg to the extent of two inches. The left hip and sacroiliac joints were ankylosed, the corresponding sacral ala undeveloped, and there was general contraction of the pelvis. The pelvic measurements were as follows:—

Anterior interspinous	7	inches.
Intercristal	8	„
Ext. conjugate	6	„
Posterior interspinous	2¾	„
Conjugata vera	2–2½	„
Transverse of outlet	2	„
Antero-posterior of outlet	4	„

The soft parts were narrow and rigid; the os admitted two fingers only, and the head lay transversely, with the occiput wedged into the pelvic brim, so that the lower uterine segment was nipped between them.

Cæsarean section was performed, the (median) uterine incision striking the anteriorly-attached placenta. No difficulty occurred in the delivery of fœtus and secundines, but, after the removal of the latter, the uterine cavity was found to be lined by a layer of greenish-yellow, pulpy material—presumably diseased decidua. It was extremely tenacious, and its removal occupied a considerable time. The uterine wound was closed by deep interrupted sutures of chromicised catgut and a superficial peritoneal layer of the same material.

On the third day there was some chest trouble which soon cleared up. But on the eighth day the temperature began to rise, and a small unhealthy sore developed over the sacrum. The lochia were normal, but, as a matter of precaution, the uterus was explored, and some more of the tenacious material was found in its lower part. After this recovery took place without further mishap.

Case IV.—Mrs. J. J., aged 28 : II gravida. (*See* Case III.) Pregnancy had commenced in July, 1901, and the patient was at once placed upon anti-syphilitic treatment, from which she derived great benefit throughout the entire period of gestation. Labour set in on the morning of April 16, 1902, and the patient entered the Lying-in Hospital some hours later. On examination the abdominal scar was perfectly sound ; the fœtus lay with its back turned to the right, and the transversely lying head was above the pelvic brim. Cervix admitted one finger ; membranes unruptured.

With the assistance of my colleague, Dr. A. Stookes, the second section was at once performed, the incision being made through the old scar, which was composed of tough cicatricial tissue ¾ inch in thickness. The perfectly smooth anterior surface of the uterus was exposed, the site of the former incision being indicated only by a faint linear depression. Adhesions were entirely absent. The uterine wall showed no trace of cicatricial tissue when incised along the depression above mentioned, and its thickness (15 mm.) no sign of diminution. The child was delivered without difficulty, and the posteriorly-lying placenta removed with the membranes. The uterine wound was closed with interrupted catgut sutures, and the uterine wall, along the site of the wound, was then sewn to the abdominal wall by seven catgut and two silk sutures. These sutures were passed through the entire thickness of the abdominal wall, so that, when they were tightened, they not only approximated the edges of the abdominal wound, but they fixed firmly to the parietes an area of the uterine wall measuring 3½ inches by 1 inch. The extreme upper and lower sutures were passed 1 cm. beyond the extremities of the uterine incision, in order that a repetition of the incision at a future section might leave the peritoneal cavity unopened and intact.

The female child was apnœic at birth, and did not attempt to breathe until after the lapse of three minutes. It weighed 6 lbs. 3 ozs.

Case V.—Mrs. B., aged 28, II gravida, patient of Dr. Humphreys, of Cressington. The patient had had one full-term labour terminated by perforation and cephalotripsy. The pelvic measurements were as follow :—

Interspinous	9¼	inches.
Intercristal	11	„
External conjugate	6⅜	„
Conjugata diagonalis	3½	„
Conjugata vera (estimated)	3	„

August 20, 1902: Dr. G. W. Little having anæsthetised the patient, I performed Cæsarean section with the assistance of Drs. Humphreys and O'Hagan. Median incision through the abdominal wall and then through uterus. The wall of the latter was unusually thick, ¾ inch. Posterior attachment of the placenta (the fœtus lay in the R.O.A.). Forefinger passed through cervix to make certain that it was sufficiently patulous for drainage. The uterus was sutured as follows:—A deep continuous layer of silk placed so as to avoid the decidua and to include two thirds of the muscular wall. Interrupted silk sutures, at intervals of an inch, brought the rest of the wall and the peritoneal coat into apposition. Utero-parietal attachment was now secured by silk sutures passing through the whole thickness of the parietal wall on each side of the incision, and also through the uterine wall to a depth of about ¼ an inch, entering and emerging the uterine wall ½ an inch on each side from the incision. The entire length of the uterine wound was so apposed to the parietes, and of the wound in the latter, there then remained 1½ inches to be separately sutured.

Uterus contracted well throughout, and bleeding was slight. Abdominal cavity was washed out with plain sterilised water before the wound was closed; this returned quite clear. Pulse at finish, 80 per minute.

Child was healthy; head unusually large.

Case VI.—Mrs. M. C., aged 25; II gravida. The patient had had a very small full-term child three years previously, but it lived only one year and four months. Subsequently, two abortions at three months occurred. Four months after the present pregnancy began, the patient consulted a medical man, who advised her to present herself at the Lying-in Hospital at once. Instead of following this advice, she saw another doctor who allowed her to go to term. On November 1, 1902, the liquor amnii escaped. A third medical man, Dr. Oldershaw, of Walton, was now sent for, and he, after anæsthetising the patient, and making a careful examination, himself brought the patient to the Lying-in Hospital. I was summoned at 1.30 a.m., when the patient was admitted. She was a slightly-built woman, 4 feet 6 inches in height, evidently rachitic. Scars of osteotomy of both tibiæ when six years of age. On examination, marked pelvic deformity was found, the measurements being as follow :—

Interspinous	9	inches.
Intercristal	9¼	,,
Bitrochanteric	11	,,
External conjugate	4⅞	,,
Conjugata diagonalis	3¼	,,
Conjugata vera (estimated)	2⅝	,,

Rachitic flat pelvis with general contraction.

The head could not be pressed into the brim of the pelvis, as marked disproportion existed between them. Cæsarean section was decided upon. There had been no pains since admission, so interference was postponed until daylight, the patient being left under close observation.

Sunday, November 2.—Assisted by my colleagues, Drs. J. E. Gemmell and A. Stookes, I performed Cæsarean section in the usual manner, making a vertical incision into the uterus. The child cried at once when delivered. The placenta was attached to the posterior wall, and occasioned some difficulty in its separation on account of firm adhesion. Loss of blood, slight. Uterus sutured with silk in two layers, and then attached to the abdominal parietes by three silkworm-gut sutures passed through the uterine wall at each extremity of the wound and in its centre. Abdominal wound closed by three layers of silk sutures, after which the silkworm-gut sutures were tightened and tied.

On November 4, a clot the size of a hen's egg was passed per vaginam, and after-pains, which up to that time had been troublesome, ceased. Uninterrupted recovery followed, and the mother left the Hospital, on November 29, with her healthy infant.

Case VII.—Mrs. M. D., aged 23. (The notes of this case have unfortunately been mislaid, so that it is not possible to give details.) The patient had a generally contracted pelvis, and had been in labour for many hours before admission to hospital. Several attempts at delivery had been made prior to this, but without success, and the patient was much exhausted when admitted. The head lay above the brim, and the disproportion between the two was very marked.

The operation was carried out in the usual way without any unusual incident. The patient appeared at first to be doing well, but symptoms of sepsis became evident, and death occurred on the fourth day.

Purulent peritonitis existed; the uterine was gaping, without attempt at healing.

Case VIII.—Mrs. W., æt. 32, married four years, patient of Dr. Doonan, Northwich. She had had one previous full-term labour (8th March, 1903), when a dead fœtus was delivered after embryotomy. The pelvic measurements were as follows:—

Sp. Il.	7¼	inches.
Cr. Il.	9	,,
Ext. Conj.	5½	,,
Bitroch.	10	,,
Conjugata diagonalis	3	,,
,, vera (estimated)	2¾	,,
Transverse of outlet	3	,,

The pelvis was generally contracted, but showed no definite signs of rickets. The patient was a little undersized woman of defective development generally. Two of her brothers suffer from Duchenne's paralysis.

Pregnancy occurred again in the latter half of 1904, and it was calculated that term would be due about 11th May, 1905. A week before that date I performed Cæsarean section with the assistance of Drs. Joynson and Doonan.

The abdominal incision was made through the sheath of the left rectus muscle. The placenta was attached anteriorly, but hæmorrhage was not great, and the child was easily delivered. The uterine wound was sewn up with three layers of continuous catgut sutures, and it was then sewn to the parietal peritoneum, a full centimetre of the uterine wall at each end of the incision being included in the attachment.

The child was feeble from the moment of delivery, and only survived a few hours. The mother recovered without a bad symptom.

Case IX.—Mrs. C., aged 28, IV gravida (*see* Case VI. for details of past labours, etc.). The last menstruation had occurred on June 4, 1904, and the patient entered the Hospital for Women on March 1, 1905, when it was considered that gestation had entered on the second half of the ninth month. The fundus uteri reached up to the xiphoid cartilage; the girth of the abdomen at the umbilicus was 34 inches. The fœtus was alive and active, the head lay transversely at the brim, but could not be pushed into it.

March 4.—Cæsarean section: the abdominal incision was made through the old median scar, and carried deeper until the uterine wall was exposed without encountering peritoneum. So far as could be observed, there was no trace of the first incision on the wall of the uterus, but, as a mere linear strip of the latter was laid bare, it is not permissible to make any definite statement. This linear strip was incised, a full half inch being cut through ere the membranes bulged, when its length was increased to four and a half inches by means of a probe-pointed bistoury guarded by the finger. The fœtal head was guided to the opening and expressed through it. Unfortunately the four-inch incision proved to be inadequate, and, during the escape of the head, a tear between $\frac{1}{2}$ and $\frac{3}{4}$ of an inch in length occurred at the lower end of the incision. In an ordinary case, this would have been a trifling incident, but in the present instance the tear opened the peritoneum to the extent of half an inch.

After the extraction of the child, citrate of ergotinin gr. $\frac{1}{100}$ was given hypodermically. The placenta, attached to the fundus, right side and part of the posterior wall of the uterus, was removed with the membrane, and the uterine wound closed. Hæmorrhage very moderate. Two layers of interrupted catgut sutures were placed, each including muscular tissue of the uterus, the inner embracing, in addition, decidua, the outer the fascial structures of the abdominal parietes. The edges of the peritoneal rent were sutured to the uterine wall. The skin was sutured separately. The child measured 20 inches in length and weighed 8 lbs. The mother made a good recovery, and left hospital with the child on April 1.

Case X.—Mrs. J. J., aged 31, III gravida (*see* Cases III. and IV.). The patient entered the Lying-in Hospital on

April 13, 1905, when it was calculated that she was within a few days of full term.

April 15.—Operation, Cæsarean section was performed before the onset of labour, Dr. Stookes assisting. The incision was made through the old scar, the tissues of which were very dense and firm. On dissecting carefully down to the uterus, it was found that utero-parietal adhesion was imperfect.

When the uterine wall was incised in the same vertical plane as the parietal incision, no thinning of the wall was apparent. The placenta was attached posteriorly, and, after its removal, the uterus failed to contract well, and free bleeding took place. Arrest was effected by manual compression and injection of ergotin, gr. $\frac{1}{100}$. Suture of the wall in three layers and then firm suture to the parietes.

After the patient was returned to bed, further hæmorrhage took place, but it was checked by a second dose of ergotin.

The child was a female, 5 lbs. 12 ozs. in weight, and showed the usual signs of maturity.

Mother and child left hospital on May 11.

Case XI.—Mrs. L., aged 38, II gravida; was sent to me by Drs. Joynson and Doonan, of Northwich. The first pregnancy (in 1902) was terminated at full term only after a difficult and prolonged embryotomy.

Marked lordosis existed, with ankylosis of the left hip joint, the left foot being 5 ins. above the ground when the patient stood on the sound leg. In the latter position, the height was 5 ft. 1¾ ins. When three years of age, the patient had fallen from a chair on to the floor. Hip-joint disease followed, an abscess formed, burst, and continued to discharge until the patient attained the age of 12 or 13 years.

The pelvis was asymmetrical, with general contraction, and approached the funnel-shaped type, the greatest contraction being on the left side. The conjugata diagonalis measured 3⅞ ins., the C.v. being estimated at 3¼ ins. The anterior-posterior diameter of the outlet was 3 ins. and the transverse 2 ins. The interspinous diameter measured 8¼ ins. and the intercristal 11 ins.

Cæsarean section at term was advised, and at once accepted by the patient and her husband, the date below being fixed as about that of full term.

May 27, 1906, operation: Dr. Joynson giving the anæsthetic, I performed Cæsarean section with the assistance of Dr. Doonan. The usual routine was followed, the placenta was attached posteriorly, and there was very little bleeding. The child weighed 11 lbs. at birth, and has thriven well since. The mother made a good recovery.

Case XII. (vide ante, Case VIII.).—Mrs. W., aged 33, III gravida. July 1, operation in the Victoria Infirmary, Northwich; Dr. Doonan gave the anæsthetic, Dr. Mainwaring-White assisting. The old scar over the centre of the left rectus was deeply pigmented, much more so than the linea alba. Incision through the old scar encountered much cicatricial tissue between skin and rectus sheath. Anterior attachment of placenta, not much bleeding.

Mother and child did well.

Case XIII.—Mrs. X., aged 38, I gravida, was seen in labour on the morning of July 19, 1906, by Dr. James Edwards. Recognising that labour was likely to be interfered with by a mass in the pelvic cavity, he asked me to see the patient. Measurement showed that the pelvis was normal, but its cavity was occupied by a swelling of boggy consistence. It entirely filled the left half, but a crescentic space an inch in breadth remained clear in front and on the right. Vagina and cervix were carried forwards behind the symphysis pubis, and upon the latter the anterior part of the fœtal head rested. Per rectum, the tumour lay in front, and its lower rounded border could be nipped by fingers introduced into rectum and vagina. The os uteri admitted two fingers: the membranes were unruptured.

The tumour was evidently a dermoid cyst of the ovary. It was quite immovable, even under anæsthesia, so we decided to open the abdomen and perform ovariotomy, if the tumour were accessible.

Operation at four in the afternoon: Dr. H. T. Nixon gave the anæsthetic, and Dr. Edwards and Mr. Scarisbrick assisted. When the abdomen had been opened, a hand was passed behind the uterus down to the tumour, but only the upper border of the latter could be touched. After several attempts, it was grasped, but no efforts could dislodge it from its position. Elevation of the uterus and its contents did not assist matters in the least ; even with the help of pressure from below the tumour could not be raised. Finally Cæsarean section was performed, the vertical incision encountering the anteriorly attached placenta. Fœtus delivered and uterus emptied with but moderate bleeding. The uterine wound was sutured in three layers with catgut, the superficial one including peritoneum only. This completed, the tumour was again attacked, but, for a time, it offered a most obstinate resistance to all attempts to displace it. However, it came up at last, and was cut away from its pedicle, which, after ligature of the arteries, was sewn up in the usual manner. The right ovary was normal.

The uterus remained contracted throughout, and the patient stood the operation well. The child, apnœic at birth, was fully developed and healthy. Mother and child have done well.

Examination of the tumour confirmed the diagnosis of dermoid cyst.

Case XIV.—Mrs. S., aged 24, II gravida. The first pregnancy was terminated after perforation in another hospital. The patient was a woman of short stature with bowing of the femora and knock-knee. There was general contraction of the pelvis, most marked anterio-posteriorly, especially on the left side. The pelvic measurements were as follow :—

Interspinous	- 8¼	inches.
Intercristal	- 9½	,,
Intertrochanteric	- 10½	,,
External conjugate	- 5¾	,,
Conjugata diagonalis	- 3¾	,,
Conjugata vera as estimated	- 3	,,
Conjugata vera actual measurement	- 3	,,

The sub-pubic arch was narrow and pointed, and the tuberosities of the ischial bones much closer together than normal, the transverse of the outlet, after repeated measurements, being estimated at 2½ inches.

Cæsarean section was advised and accepted by the patient and her husband.

Menstruation had occurred last on November 7, 1905, but there was a very short period at the end of December. It was, therefore, arranged that the patient should enter the Lying-in Hospital on August 14, 1906.

On admission, I judged that full term was still two or three weeks off, so the patient returned home. She re-entered hospital on August 30, and, on the afternoon of September 2, experienced intermittent abdominal pains. Being out of town, I did not see her until 10.30 p.m., when it was found that the external os admitted a finger, and that the internal os and cervical canal were dilating.

11.20 p.m.—Operation. Dr. S. L. Harke administered chloroform and ether, and my colleague, Dr. A. Stookes, assisted me. There was more bleeding from the uterine incision than usual, in spite of the posterior placental insertion, but the blood escaped principally from two large veins, its flow ceasing after the extraction of the fœtus. The latter appeared to be in the apnœic state when delivered, although the heart was beating but slowly. After waiting a little time, the heart-beats became more feeble, and no attempt was made at respiration. The cord was at once divided, and Dr. Murray Cairns kindly took charge of the fœtus, the ultimate recovery of which was entirely due to his unremitting care and perseverance.

The membranes were firmly attached to the uterus, and some time was occupied in peeling them off. Then a little trouble occurred with the anæsthetic. Beyond these incidents the operation was uneventful. Convalescence progressed normally until the evening of the fifth day, when the temperature rose to 103°. The pulse rate was 110 and the general condition, one of well-being. However, an intra-uterine douche was given half a drachm of Tr. iodi to the pint of sterilised water being used. The solution returned turbid, but it contained no clots. Next morning the temperature was 100°, and the douche was repeated. Three hours later a rigor occurred, the temperature rising to 105°. Douching was discontinued, and the temperature speedily returned to normal, the pulse rate subsequently remaining below 90. On two subsequent evenings, the temperature was just above 100°, and on one occasion 101°·6.

The child made but slow progress for the first fortnight; from that time onwards it thrived well, and was in good condition when the patient was discharged on October 11th. Examination made before discharge ascertained that the uterus lay high up, quite adherent to the abdominal wall.

Case XV.—Mrs. W., aged 26, I gravida. The patient measured 3 feet 6 inches in height, and was a typical specimen of the achondroplasiac dwarf. She was married in February, 1900, and stated that the last menstrual period had occurred in March, and that she considered herself to be seven months pregnant. In spite of her diminutive stature, she had never had any misgivings regarding her confinement, and did not think it necessary to see a doctor until labour actually began. Labour pains set in on the evening of Saturday, September 15th, and Dr. Hutchinson of Widnes saw the patient at 11 p.m. He at once recognised that delivery *per vias naturales* was impossible, and sent the patient by the first available train to the Lying-in Hospital.

The pelvic diameters were as follow :—

Interspinous	6¾	inches.
Intercristal	8	,,
External conjugate	5½	,,
Conjugata diagonalis	2½	,,
Conjugata vera (estimated)	1¾	,,
,, ,, (measured)	2	,,

The fundus uteri lay 1½ inches below the xiphoid cartilage ; the head lay upon the upper border of the symphysis pubic, bulging the abdominal parietes forward, and forming a swelling plainly visible on inspection. The fœtus lay with its back to the front and to the right : fœtal heart sounds audible. The uterus was contracting at intervals. Per vaginam : the cervix lay high up immediately behind the pubes, and was reached with difficulty : the external os admitted two fingers which felt the bag of membranes lying in the cervical canal.

It was evident that the pregnancy was but a month short of full term, and also that the only possible method of delivery was by Cæsarean section.

Sunday, September 16th : operation. Dr. Bertram Smith gave the anæsthetic, and my colleague, Dr. J. E. Gemmell, assisted me.

The high position of the uterus necessitated the placing of the parietal incision at a higher level than usual, its upper extremity being just above the umbilicus. It was made as usual through the sheath of the left rectus abdominis muscle. The incision through the uterine wall was followed by considerable bleeding, the placenta being inserted anteriorly. Otherwise there was nothing of note about the operation.

My colleague, Dr. Murray Cairns, took charge of the child, which breathed soon after extraction. It measured 19 inches in length, and weighed 5 lbs. 3 ozs.

The mother made an uninterrupted recovery, and left hospital with her child on October 18th.

Case XVI.—Mrs. B. S. (German Jewess), aged 26, III gravida. The patient arrived in this country at Christmas, 1905, at which time the last menstrual period occurred. There had been two previous full term labours, both difficult and terminated by forceps, a dead child being extracted on each occasion.

The patient was an undersized woman, her height being 4 feet 9¾ inches. The pelvic measurements were as follow :—

Interspinous - - - -	9	inches.
Intercristal - - - -	10	„
Conjugata externalis - - -	6	„
Conjugata diagonalis - - -	3¾	„
Conjugata vera, estimated - - -	3	„
Transverse of outlet estimated as about	3¼	„

There were no signs of rickets having existed in childhood.

The deformity was explained to the patient two months before labour full term was due, and the possible methods of treatment made clear. Her great anxiety was to obtain a living child, and of her own accord she selected the section at full term.

Labour pains set in about 8 p.m. on September 23rd. The head lay above the brim, and the disproportion between the two was very marked.

Later in the evening operation was carried out, Dr. Edmiston acting as anæsthetist, and Dr. J. E. Gemmell assisting me. Hæmorrhage was slight, the placenta being attached to the posterior wall and fundus of the uterus. The membranes were adherent, and considerable care was needed in their detachment ; the placenta came away without difficulty.

The child was separated 2½ minutes after birth. It had not breathed, but the heart was pulsating strongly. The child was immersed in a hot bath, but respiration did not take place satisfactorily until Schultze's method of artificial respiration had been practised for 30 seconds. Respiration was shallow for five minutes, but improved after vigorous crying. The child measured 19½ inches in length, and weighed 7 lbs. 7 ozs.

Convalescence progressed normally, mother and child leaving hospital a month after operation.

The Parietal Incision.—In the case of the first six patients, the median vertical incision was employed. Since 1902, the extra-median incision through the sheath of the rectus abdominis has been used in all my laparotomies, but I hesitated at first to apply it in cases of Cæsarean section. Experience of it, in Case VIII., disclosed no disadvantages, so it has been used in all the remaining cases.

With regard to the length of the incision, it will be sufficient to quote Sir William Sinclair's words[1]:—"I have read and heard that the wound need not be more than four inches in length. As the true conjugate of the pelvis is more than that, it may be inferred that a four-inch incision is an estimate, not a measurement as applied to the uterus in Cæsarean section. An added half inch or more of a clean incision is a small matter compared with laceration of the uterus or asphyxia of the fœtus."

Eventration of the Uterus (*i.e.*, drawing it forward and out of the abdomen) was employed in the first case only, simply because it was a first case, and the control of hæmorrhage seemed an easier matter when the uterus was lying outside instead of within the abdomen. But there was no bleeding in that case, and so eventration has not been practised in the other fifteen. Not only is it unnecessary, but its use requires a long parietal incision, with increased after-possibility of hernia. Further, despite all the care that may be taken to keep an eventrated uterus warm, moist and free from bruising, there can be no doubt that drying and bruising of its peritoneal coat do occur. This is shown in the records of some cases of repeated Cæsarean section on the same patients, in which there have been found adhesions between intestine or omentum and parts of the uterine wall remote from the wound. Only peritoneal traumatism can explain the formation of such adhesions. The peritoneum of a uterus, maintained within the abdomen, cannot become dried by exposure to air, and, as the abdominal parietes intervene between it and the hands of the assistant, bruising is unlikely, much less probable than when the only buffer is a warm cloth or towel. Skilfully applied pressure can prevent the influx of blood, etc., into the cœlom, and can usually control bleeding from the uterus. Should hæmorrhage be excessive, after extraction of fœtus and secundines, the four and a half or five inch parietal wound is quite long enough to permit immediate extrusion of the uterus. This can be done also should any difficulty arise in the suture of the uterine incision.

It would seem advisable to limit the use of eventration to those cases in which the existence of sepsis is suspected, and in which the intention is to terminate the operation by Porro's modification.

Diagnosis of the Placental Site.—In pre-antiseptic times, great attention was paid to this point, since wounding of the placental site was held not only to incur risks of hæmorrhage but to increase the danger of sepsis. At the present day, hæmorrhage is practically the sole consideration, and although the bleeding is usually moderate, yet it may be very profuse. Therefore, Olshausen, whose experience in Cæsarean section is surpassed by but few operators, emphasises strongly the advisability of making the uterine incision so as to avoid the placental site, and urges the importance of placing it anywhere *along the median line*, provided that the thickened wall, corresponding to the placental attachment, is not invaded. Sippel, another operator, actually opens the uterus through its posterior wall when an anterior placental insertion is recognised. But, after all, to encounter the placenta does not amount to a catastrophe ; the bleeding is usually quite moderate, and practically ceases with the extraction of the fœtus. Some comfort, however, may be afforded to the mind of the operator, if he knows definitely that his incision is, or is not, going to encounter the placenta. In some cases, the diagnosis cannot be made, but in the majority, it is possible with tolerable certainty to recognise the site of the placental attachment to the uterus ere the latter be opened.

Several points[1] assist in making this diagnosis. It is stated that, under normal conditions, the ventral aspect of the fœtus lies opposite to the placental site, so that an anterior position of the fœtal back should arouse expectation of a posteriorly attached placenta. Murdoch Cameron[2] pointed out this relation in 1896, but his investigations were carried out in a limited number of cases, and the results do not harmonise with those obtained by Caruso.[3] The latter made observations on about 1,000 cases, and from them concluded that anterior insertion was by far the commonest. It is necessary also to bear in mind that pelvic deformity (the usual indication for the operation) may modify the fœtal position.

In my experience, the most trustworthy diagnostic method lies in comparison of the superficial areas of anterior and posterior walls of the uterus, but more especially of their widths, as estimated between the insertions of the Fallopian tubes and round ligaments. For example, if the placenta is attached chiefly, or entirely to the anterior wall, the latter will attain a superficial area exceeding that of the posterior wall, and the difference will be particularly well marked in the widths. This is clearly visible when the pregnant uterus is eventrated, but when it still lies in the abdomen, a hand introduced within the abdominal cavity can readily distinguish the difference by palpation. If it exists at all, the difference is well enough marked to remove all doubt from one's mind.

Further, the exploring hand may detect distinct bulging over some part of the uterine wall : such a bulging area of soft consistence invariably implies an underlying placenta, but its occurrence does not appear to be very common. In but one of my cases was it found, in Case XVI., in which the posterior wall was much broader than the anterior, and the whole posterior aspect of the fundus bulged sharply up from the posterior wall and the anterior half part of the fundus, almost as though some tumour of the uterus existed. The placenta, as was anticipated, was found to be attached partly to the posterior wall and partly to the fundus.

Palm[4] has pointed out that when the uterus approaches somewhat to a spindle form, the placental insertion is low, and conversely it is high with a more spherical uterus.

Finally, it has been stated that the presence of numerous visible vessels, especially of venous networks, on the anterior wall, indicates an anterior insertion. It may do so in some cases, and, when present, may be taken as corroboration of some other sign with which it is in harmony, but, in the only instance in which I have seen it well marked, the placenta was inserted posteriorly.

Opening the Uterus.—The vertical median incision through the anterior wall was employed in preference to the "Fundalschnitt," for two reasons :—

First, eventration is unnecessary.

Secondly, with its use the operation can be completed by the formation of a definite utero-parietal adhesion between the anterior wall of the uterus and the abdominal parietes. This utero-parietal adhesion serves two purposes, it prevents the formation of haphazard, and sometimes dangerous, adhesions between the uterus and other viscera, and its existence in a sufficiently extensive form may permit the repeated performance of Cæsarean section, at a future date, without the opening of the peritoneal cavity. In a case of perfect utero-parietal adhesion, the incision would pass from the parietes directly into the uterus. When the fundal incision is used, such perfect utero-parietal adhesion is impossible.

The advantages of the median vertical over the transverse incision will be seen from the subjoined tabular arrangement of arguments for and against the latter :—

"FUNDALSCHNITT."

Arguments for :—	Arguments against :—
The course of the incision is parallel to that of the vessels, therefore bleeding, especially that from spouting arteries, is at once checked by the application and tightening of sutures.	In the middle line spouting arteries are rarely found—Olshausen has never seen them. The bleeding is purely venous and can be checked by pressure.
Easy extraction of the fœtus - -	There is no difficulty in extracting the fœtus through the vertical incision, if it is large enough.
Marked and rapid diminution of the wound.	—
Utero-parietal adhesion cannot occur, as the uterine and parietal wounds do not lie in contact.	This is a drawback, because such adhesion is a desideratum when it takes place in the proper manner. But, utero-intestinal adhesions have occurred, and in two cases utero-parietal fistula. Both of these are serious conditions.
The abdominal incision can be made much higher above the pubes, thus lessening the liability to incisional hernia.	An incision, made through the sheath of the rectus muscle, practically diminishes the risk of hernia to zero. Olshausen points out that, with the vertical incision, the parietal wound need not approach nearer the pubes than 6–8 cm., "just enough to allow of eventration:" the same holds good for the "Fundalschnitt."
	Eventration is necessary for its performance.

When it is recognised that the placenta is inserted anteriorly, some operators plan the sagittal incision so as to avoid its site. Sippel, for example, has twice cut through the posterior wall of the uterus. Olshausen makes a small incision into the uterine cavity, and then, having determined by vision or palpation the precise situation of the placenta, prolongs the incision away from its edge, over the fundus and on to the posterior wall, if necessary. (Olshausen, like most German operators, always eventrates the uterus.) When it seems certain that the placenta is attached anteriorly, my own plan is to mark out, by a superficial cut, the full length of the incision, to deepen the middle part until the placenta is reached, and then to rapidly complete the wound by means of scissors (or a probe-pointed bistoury) guarded by the finger. The placenta is then separated *towards* the cervix, for, in this direction, the operator will soonest find the placental edge and the commencement of the membranes.

Hæmorrhage during Operation.—In no case was there bleeding of a serious kind, that is, a loss free enough to cause any anxiety. Hæmorrhage may occur from

(1) the uterine incision;

(2) the placental site during separation of an anteriorly detached placenta;

(3) the placental site after delivery.

There is rarely much bleeding from the uterine incision, unless the placenta is anterior. In one case (XIV.), in which the placenta was posterior, the incision bled freely from two large sinuses in the uterine wall, but it was readily checked by pressure, and ceased as soon as the fœtus was extracted.

When an anterior placental insertion complicates the operation, the more vascular anterior wall may bleed freely, and the stripping off of the placenta set up further loss. But the hæmorrhage in such cases is sometimes astonishingly slight: in six of the cases here recorded, the placental insertion was anterior, but in two only did the hæmorrhage exceed the usual, whilst it was strikingly small in the others.

In the control of bleeding, the hands of the assistant play the most important part, by judiciously applying pressure With a lax abdominal wall, a skilful assistant can reduce the bleeding to a minimum. But, in addition, there should be rapid emptying of the uterus, and quick closure of its wound.

Usually, a hypodermic injection of ergotinin citrate, gr. $\frac{1}{100}$, was given after the extraction of the fœtus, as a matter of precaution merely; most of the cases would have done quite well without it.

Many courses have been recommended for dealing with severe hæmorrhage, and massage of the uterus, compression of the broad ligaments manually, or by means of a rubber band, even hysterectomy, have all been advocated and tried. Murdoch Cameron's extensive experience led him to believe that hæmorrhage was best overcome by wrapping the uterus in a warm flat sponge and compressing it, and he stated "in no case has the uterus failed to respond to this method of compression."

According to some writers the matter of hæmorrhage has been connected with another question, that of the *time to operate*. Is it better to operate by daylight, at a fixed hour, before the commencement of labour, or to await its onset?

In nine instances, operation was undertaken during labour; in seven before its commencement. In six of the latter, the bleeding was not unusual; in one, it was profuse, the uterus contracting imperfectly. Of the nine cases operated on during the progress of labour, in six, the ordinary amount was lost; in the other three, the loss exceeded the usual, but in two of these, there was an anterior placental attachment, and the hæmorrhage came principally from the incision through the vascular uterine wall. In one case, labour had been in progress, more or less, for sixty hours before operation, but very little bleeding occurred during its performance. On the other hand, the greatest loss of blood took place in a patient operated upon before the onset of labour.

Olshausen speaks of the "great probability" of bleeding in such a case, and advises waiting until some dilatation of the cervix has taken place. It is reasonable to expect good uterine contraction and retraction in cases operated on after labour has been initiated, but one can hardly anticipate equally good results from a uterus surprised and stormed ere the rhythmical swing of contraction has become established. If either course offered a distinct advantage to those mainly concerned, the mother and the fœtus, the choice would be easy. But the truth would appear to be that each has its own drawbacks, and, to my thinking, there is less disadvantage in operating by daylight at a fixed hour, after full preparation, than there is in leaving the time to chance on account of possible hæmorrhage that rarely occurs.

Case.	Operation before or during Labour.	Placental Insertion.	Hæmorrhage at Operation.	Postpartum Contraction of Uterus.
1	Before onset - -	Posterior -	Slight - -	Porro's operation.
2	Labour in progress	Anterior -	,, - -	Good.
3	,, ,, ,,	,, -	,, - -	,,
4	,, ,, ,,	Posterior -	,, - -	,,
5	Before onset - -	,, -	,, - -	,,
6	Labour in progress	,, -	,, - -	,,
7	,, ,, ,,	—	—	,,
8	Before onset - -	Anterior -	Slight - -	,,
9	,, ,, - -	Posterior -	Very moderate	Fair.
10	,, ,, - -	,, -	Free - -	Bad.
11	,, ,, - -	,, -	Very little -	Good
12	,, ,, - -	Anterior -	Slight - -	,,
13	Labour in progress	,, -	Moderate -	Fair.
14	,, ,, ,,	Posterior -	*Moderate -	Good.
15	,, ,, ,,	Anterior -	*Moderate -	,,
16	,, ,, ,,	Posterior -	Slight - -	Fair.

* Bleeding from uterine incision.

Suture of the Uterus.—This part of the operation was always omitted by the earlier operators, and yet an astonishing number of their patients recovered, sometimes with persistent utero-parietal fistulæ as mementoes of their escapes. The resulting cicatrix was not a substantial one, and it was not always strong enough to withstand the strain imposed by subsequent pregnancy. This giving way of the scar is a danger which, even at the present time, pursues the Cæsarean operation. When the child-bearing capacity of a patient is destroyed by some so-called sterilising procedure, suture of the wound does not require more than ordinary care. But, when the operation is carried out, as it should be, with a view to future pregnancy, then the most perfect apposition of the wound surfaces is demanded. They must be brought accurately together from decidua to peritoneum in order that exact union may occur with a minimum formation of cicatricial tissue. Not mere healing, but total obliteration of all traces of the wound, should be aimed at, and that this is no mere visionary quest the records of repeated sections show, for they not infrequently state that not only was the uterine wall of normal thickness, but that it was difficult to recognise where the earlier incision had been made.

It must be remembered that, after the emptying of the uterus, the length of the incision has diminished, while its depth has increased in consequence of the thickening of the uterine wall. Raw surfaces, varying from three-quarters of an inch or more, have to be perfectly apposed, and, for this coaptation, it is frequently stated that two layers of sutures are necessary. I think three at least are required for perfect union, and, with a thick-walled uterus, four are advisable. In my earlier cases, two layers only were employed and with good result, but it is better to err on the safe side, and, if by reason of a multiplicity of sutures, the wound site is thicker than the rest of the uterine wall, there is all the greater prospect that the involuted uterus will possess a uniformly thick wall capable of successfully withstanding the stretching caused by a later pregnancy.

The deepest (first) of the three layers of sutures is made with a continuous catgut suture that includes decidua and part of the muscular wall. Part of each stitch lies exposed in the uterine cavity, and the suture material must, therefore, be one that is capable of becoming easily absorbed. The second layer includes muscle only, and the third, muscle and peritoneum. Universal experience has shown that whether silk or catgut be employed for these layers is a matter of indifference, provided the material be sterile. Silkworm gut has been used without any untoward result. In one of my repeated operations, a piece of silkworm-gut suture was found buried in the uterine wall, in which by error it had been sewn.

Utero-parietal Suture.—It has too long been the custom to perform Cæsarean section without making provision for the future safe repetition of the operation. On the contrary, many operators have rendered further pregnancy impossible by occluding Fallopian tubes, or removing ovaries, or even uterus. Of recent years, this procedure has met with opposition, led in this country, by W. J. Sinclair, H. R. Spencer, and, latterly, Munro Kerr, with the result that it is now becoming recognised that removal of healthy reproductive organs, or destruction of their functions, are matters which lie entirely outside the domain of the obstetrician. But, so long as the child-bearing capacity is preserved, it *is* the business of the obstetrician to endeavour to take steps, which will render subsequent repetitions of the operation safer for the patient, and easier of performance for the operator.

At the performance of the second or subsequent operation, it is found that, in the majority of cases, the operation is complicated by the presence of more or less troublesome adhesions which have formed as the result of the first section. These occur between uterus, intestine, omentum, and abdominal wall, any two or three, or even all four, becoming united by adhesions of varying density and extent. Despite all attempts to prevent them, they continue to appear at the repeated operation.* In a few cases, there has been recorded complete adhesion of the anterior uterine wall to the anterior abdominal parietes, with the result that the section has been carried out without opening the peritoneal cavity. Under such a condition, the operation is robbed of some of its risks, since it resolves itself into a simple incision from skin to decidua, whilst troublesome intestinal adhesions are not encountered. To bring about such a desirable state of matters, W. J. Sinclair endeavoured to produce utero-parietal adhesion at the first operation, by rubbing iodoform over the anterior uterine wall, and firmly bandaging the abdomen after its closure. I suggested, in 1902, that the adhesion might be produced by sewing the uterus to the abdominal wound, and, since that time, have carried this out in every case, in which the indication for operation has been pelvic deformity. When the uterine wound is closed, it is sewn to the parietal incision, the highest and lowest sutures including uterine wall 1 cm. above and below the extremities of the wound. The uterus is thus anchored to the middle line, and there is prevented that lateral movement which would expose both sutured wounds to the contact of the abdominal viscera.

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* For a full account of adhesions, *see* the author's paper "On Repeated Cæsarean Section," *Jour. Obstet. and Gynæcol. of Brit. Emp.*, 1902, Vol. II.

Further, by placing a flat sandbag over the dressing, the abdominal wall is firmly pressed down upon the underlying anterior wall of the uterus, so that when the patient strains there is less chance of intestine or omentum being forced between the two and coming into contact with lymph effused around the line of the uniting sutures.

Mortality.—Of the 16 operations, one resulted fatally to the mother, so that the maternal mortality is 6·25 per cent.

All the children were delivered alive, but one survived only a few hours. Two died from infantile maladies after they had passed from observation. The others, to the best of my knowledge, are alive and well.

The maternal fatality was due to sepsis. The patient had been in labour for hours, and several attempts at delivery had been made before she was admitted to the Lying-in Hospital. The child was alive, and, in view of the pelvic deformity, the only possible course was to perform Cæsarean section. This was carried out with the usual precautions, but with the result stated.

In a case, in which repeated serious attempts at delivery have been made, before the possible necessity for Cæsarean section has been considered, it is a question for discussion whether the operation should be terminated by Porro's modification. Prolonged manipulation may imply infection of the vagina and even of the uterus, and, in either case, a hysterectomy would lessen the risk by removing the absorptive surfaces.

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POST-PARTUM HÆMORRHAGE.

By E. HASTINGS TWEEDY, F.R.C.P.I.,
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I HAVE no desire to enter into the controversy that is at present taking place in respect to the treatment of post-partum hæmorrhage.

Dr. Gordon Fitzgerald's extensive midwifery experience entitles him to speak with considerable authority, and the methods which he recommends may with safety be left to his advocacy.

I should, however, be lacking in bravery did I permit some statements of Dr. E. Stanmore Bishop to go unnoticed. He writes :—

"That post-partum hæmorrhage is essentially a general practitioner's tragedy. I have never seen a case in hospital, and one or two other consultants, with much larger experience than mine of hospitals for women, have assured me that they, too, have never seen such cases in hospital practice."

I cannot think that these statements emanate from men who reside in the maternity hospitals to which they are attached, for in no other way can I account for their belief in the rarity of this condition amongst hospital patients.

In looking up the Rotunda statistics for the three years ending November 1, 1906, I find that amongst 5,695 women delivered within the hospital wards there occurred 56 cases of post-partum hæmorrhage. Of these no less than 36 were associated with mechanical causes which prevented the closing down of the uterus, such as retained placenta, membranes, or blood-clots ; three were traumatic in origin, whilst four were of the secondary variety. Three patients died of supervening shock.[1]

Pressure exerted on the abdominal aorta, as a means of temporarily controlling hæmorrhage, is a method which has been practised in the Rotunda for many years. It is mentioned in Jellett's *Midwifery*, and also in the first edition of Lusk.

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[1] These three deaths will shortly be published in my Hospital Report for year 1905–6.

Personally I have over and over again availed myself of it as a temporary and valuable expedient.

Dr. Stanmore Bishop attributes to Dr. Fitzgerald the discovery that the retraction force of the uterus is quite independent of contraction. I have consistently taught this since taking up my present position, but the credit of the discovery lies neither with Dr. Fitzgerald nor myself, but was enunciated some four years ago by a British writer, whose name has escaped my memory.

In favour of his view I may be permitted to adduce the following arguments.

The uterus does not commonly contract for seven minutes after the birth of the child. If the cessation of hæmorrhage depended on contraction, women should flood freely during this time, and start again to bleed after the contraction passes off.

If it is claimed that the contraction, which brings about the birth of the child, is a cause of the saving retraction, my reply is, that a woman who has been delivered by forceps without the onset of labour contractions must then inevitably bleed to death, and we know that this is not the case.

But the clearest proof that I can furnish of the interdependence of these two forces is that afforded during the operation of Cæsarean section.

Whilst stitching the uterus up after the extraction of the child, I have never failed to note and demonstrate the progressive thickening of the uterine walls, and this despite the absence of any contracting force.

Slight mechanical causes seem capable of interfering with retraction, and this appears to be the reason why a full bladder, or piece of retained placenta, will promote the occurrence of hæmorrhage, notwithstanding the fact of the uterus responding by contractions to stimulation.

SOME POINTS CONNECTED WITH EMBOLISM.

A CLINICAL LECTURE.

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[With Plates VI. and VII.]

In a vast collection of living cells, such as the human body, the problem of providing food for each individual cell is necessarily a complex one. A living cell feeds by taking into itself something from without. This process is easily and simply accomplished, where a single cell forms the whole individual, as in *Amœba.* Its whole surface is always in contact with material external to itself. All it has to do is to take into itself, or "feed" upon, such suitable material as may come into contact with it. But, in a multicellular organism, such as the human body, there are millions of cells, each of which requires to feed, and it is obvious that were the same simple arrangement to hold good as suffices for *Amœba*, only those cells which were fortunate enough to lie on the surface of the mass would come in contact with external material to feed upon, the remainder must feed upon each other or die of inanition.

This difficulty is got over by a system of irrigation, by liquid-containing canals, which we call the circulating system. The ports of entry to these canals are, for the sake of convenience, placed within the body instead of outside, viz., in the digestive tube. From these ports of entry, or docks, suitable material, in a liquid or soluble form, is distributed by the circulatory canals to every cell of the body.

Thus, by an elaborate "water-carriage" system, each individual cell receives at its own cell surface "external material" upon which it can feed as freely as if it were a unicellular individual.

Thus we see that, from a mechanical point of view, the circulatory system is merely an arrangement of pipes, or tubes containing a liquid medium, which is kept moving by certain forces to which we need not here refer.

But, like other elaborate arrangements, the circulatory system, from its very complexity, is liable to get out of order in many ways, and it is to one of these, viz., embolism, or embolic plugging, that I wish to refer.

By embolism, we mean the plugging, or obstruction, of a blood-vessel by solid material, carried by the blood-stream from some other part of the circulation. In ordinary life, such solid material cannot get into the circulation from without, it must, therefore, be formed within the vessels themselves. That such a thing can happen is due primarily to that property of the blood which we know as thrombosis, or clotting within the living blood-vessels. There is, however, another factor, a purely mechanical one, which is essential for embolism, that is the constant variation in diameter of the blood-vessels along the course of the blood-stream. Were all the blood-vessels of uniform diameter, embolism would be impossible.

I purposely refrain from entering into any discussion as to the morbid conditions which set up this act of solidification, or thrombosis, but regard them as assumed in order to centre attention upon the problem of embolism itself.

As we have seen, the liability to embolism is a necessary defect of the circulatory system, and is inseparable from the existing vascular arrangements of the organism.

Its occurrence always implies a pre-existing morbid condition or conditions capable of producing something in the blood-stream of such a size or shape that it cannot freely pass through all the vascular channels. In short, it cannot circulate.

As soon as this "something" reaches a point in the circulation at which, owing to the gradually diminishing calibre of the successive vascular branches, it is too large to go further, the act of embolism occurs.

It is obvious that the exact position in the circulation at which, in any individual case, embolism occurs, depends upon the size of the embolus. This must be larger than the largest blood corpuscle, and may be of any diameter from that upwards to that of the widest blood-vessel. It is also obvious that, speaking generally, embolism can only occur in places where the blood-stream is running from larger to smaller tubes. For this reason, the circulation, from the point of view of embolism, may be divided into three distinct parts, separated from each other by impassable capillaries. One part extends from systemic venules to pulmonary arterioles, another from pulmonary venules to systemic arterioles; a third from gastro-intestinal venules to interlobular portal venules.

Each length is bounded, both up stream and down stream, by capillaries, so that no plug sufficient to block a vessel can pass from one set to another. Occasionally there is an exception in that a patent *foramen ovale* allows an embolus from the right heart system to pass into the systemic arteries instead of into the pulmonary arteries (paradoxical embolism).

Of the three sites of embolism mentioned above, the third, viz., the portal system, is not of equal importance to the other two, for reasons which need not detain us now. We may, therefore, limit our attention to the first two, namely, the systemic and the pulmonary arteries. Each of these draws its emboli from a distinct source, as may be seen from Fig. 1.

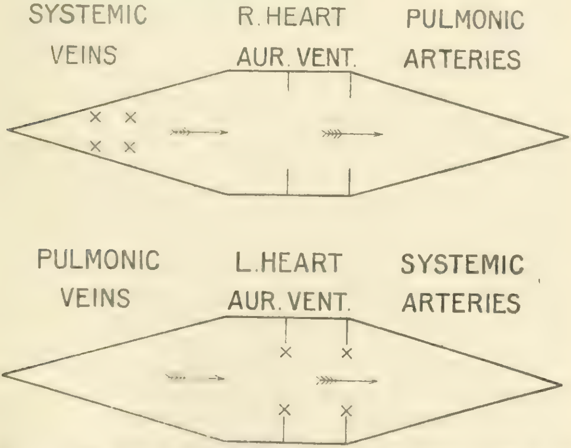

Fig. 1.—*Diagrams to illustrate the two distinct embolism-systems in the circulation. Arrows indicate the direction of current. The positions marked × × show the common sites of origin of emboli in each system.*

I have referred to this point at some length, because I know that confusion does not infrequently exist, as to the distinction between pulmonary embolism and systemic embolism, and that such confusion is not entirely limited to students.

The act of embolism is in itself, then, a simple physical phenomenon ; its results, whether immediate or remote, are much more complex, and involve the consideration of many chemical, bacteriological, and physiological factors.

What may or will happen when embolism has occurred is extremely variable. It may cause no symptoms, or it may cause profound ones. It may cause the death of a part, or it may cause the death of the whole organism.

I propose to limit my remarks to the consideration of two types of embolism, of which I have excellent specimens with which to illustrate them.

MULTIPLE EMBOLIC ANEURYSMS.

It is generally recognised that cerebral hæmorrhage in children, apart from any general disease such as purpura, etc., is commonly due to the rupture of an intracranial aneurysm produced by an embolus in the form of a valvular vegetation from the left heart.

In this case (Fig. 2), the child, a girl, aged 12, suffered from rheumatic fever, two years before with endocarditis. She was readmitted into hospital, in July 1904, with symptoms of infective endocarditis, vomiting, headache, anæmia, hectic temperature, dyspnœa, frequent pulse, and loud aortic and mitral murmurs. One month later, the pyrexia increased, and she had a right hemiplegia with aphasia. She was not unconscious. One month later, she was suddenly seized with convulsions of the right side of the face and right arm, and rigidity of the other limbs. She became comatose, and died the same day.

At the autopsy, extensive valvular endocarditis was found, with old simple infarcts in the spleen and kidneys. A large hæmorrhage (Fig. 2) was found ploughing up the left hemisphere very extensively, and rupturing into the lateral ventricle and from there into the third and fourth ventricles also.

It is probable that the original hemiplegia with aphasia, but without loss of consciousness, which occurred four weeks before death, was due to the mechanical effect of the embolus itself plugging a cerebral artery. Had the embolus been simple, that is aseptic, no further changes, save those of degeneration from impaired blood supply, should have followed. But the organisms contained in the embolus, set up, at its resting place, an infective arteritis, leading to softening of the vessel wall, aneurysm and, eventually, rupture. This bursting was so extensive as to cause fatal results within a few hours.

That is the theory which anyone might have offered from a study of the history and the post-mortem findings in this case. Fortunately, however, the details of the process, lost in the large and destructive cerebral hæmorrhage, are demonstrated for us in this specimen (Fig. 3), which was obtained from the same case in another part of the body.

On removing the abdominal viscera, a small swelling was seen situated on the left side of the brim of the pelvis. It was hemispherical, measuring about one inch in diameter, and looked like a hæmorrhage under the peritoneum. On cutting through the peritoneal covering, a quantity of blood clot and débris was found lying over the lower end of the left internal iliac artery. The lesion, together with the tissues around, was removed, and on being carefully dissected out the appearances seen in this specimen were found (Fig. 3). In the upper part is the lower end of the left internal iliac artery, and, at an interval from this, are seen the various terminal branches it gives off. On the outer side, is the internal iliac vein, through which a white glass rod has been passed. The hæmorrhagic swelling, seen at the autopsy, lay over the position of the gap at the lower end of the artery, where the walls of the vessel had become completely softened and had given way. At the extreme lower end of the internal iliac artery (upper portion in Fig. 3), and, at the upper part of the lower fragment, the intima is seen to be thickened and covered with rough thrombus. Here, then, is a ruptured artery, the walls of which have been completely severed across in their whole circumference. There can be no doubt as to this having been primarily caused by an embolus, because, on the one hand, there were numerous evidences of emboli in many other places, brain, spleen, and kidneys, and, on the other hand, the site, namely, the giving off of numerous much smaller terminal branches from a larger trunk, is exactly such as to suit the requirements of embolism.

This embolus, being not a simple or aseptic embolus, which would merely set up local thrombosis in the immediately neighbouring parts of the blood channels, but, containing active organisms, has caused an acute local arteritis around itself.

This has produced dilatation of the artery at this point, probably as a fusiform aneurysm, and eventually complete destruction of the vessel walls, so that the blood has passed through into the neighbouring tissues. In this process of disintegration, the embolus has itself become softened and indistinguishable from the products of its activity. The small extent of the hæmorrhage suggests that this rupture had probably occurred only very shortly before death. It was formerly supposed that such an embolic aneurysm was produced behind the embolus by the extra pressure in the vessel due to the plugging, but, as was pointed out many years ago, if that were so, similar aneurysms should follow an ordinary ligature.

It is impossible now to discuss the relationship of simple rheumatic endocarditis to so-called infective endocarditis, but it is by no means uncommon to find, as in this case, an endocarditis, which begins with an ordinary attack of rheumatic fever, take on sooner or later an infective character, and rapidly prove fatal on that account.

FATAL PULMONARY EMBOLISM.

The next series of specimens illustrate the more immediately fatal effects of simple emboli in the other system, namely, the pulmonary. Whilst emboli, in the systemic arteries, whether simple or infective, almost always arise from vegetations on the valves of the left heart, those of the pulmonary arteries usually start as thrombi in the systemic veins. As such venous thrombosis, once started, readily extends from smaller to larger veins, and, as it is always that part of the thrombus nearest to the right heart in the circulation (*i.e.*, the largest end), which is washed off and forms the embolus, it is not uncommon for very thick and very long emboli to be found in the pulmonary arteries.

These specimens (Figs. 4 and 5) of embolism of the pulmonary artery itself have certain features in common, and yet certain differences, as will be seen from their histories.

The history of this case (Fig. 4) is as follows:—The patient, who was under the care of my colleague, Mr. Lockwood, to whom I am indebted for permission to use it, was an elderly man, suffering from complete obstruction of urine due to enlarged prostate. No catheter could be got in, and relief was obtained by supra-pubic puncture. After this, catheterisa-

PLATE VI.

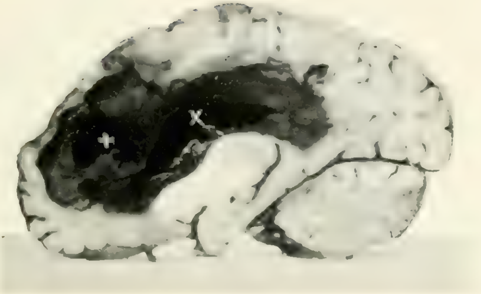

Fig. 2.—V.S. through left brain, seen from without. Cerebral hæmorrhage in girl, due to embolism. + Site of rupture. × Lateral ventricle distended with blood.

(From the Pathological Museum, University of Sheffield.)

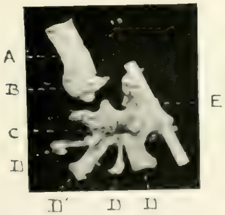

Fig. [illegible]—*Embolic softening of left internal iliac artery, causing rupture. A. Trunk of left internal iliac artery. B. Thickened infiltrated surface. C. Same as B. D, D′, etc. Terminal branches of internal iliac artery. E. Internal iliac vein.*

(N.B.—*B and C indicate the limits of the hæmorrhage.*)

(From the Pathological Museum, University of Sheffield.)

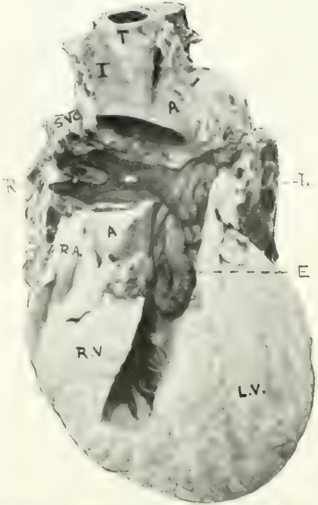

Fig. 4. *Pulmonary embolus in situ.* A A. [illegible] *I. Innominate artery. T. Trachea. R. Root of right lung. L. Root of left lung. R.A. Right auricle. R.V. Right ventricle. L.V. Left ventricle. S.V.C. Superior vena cava. E. Lower end of embolus.*

(From the Pathological Museum, University of Sheffield.)

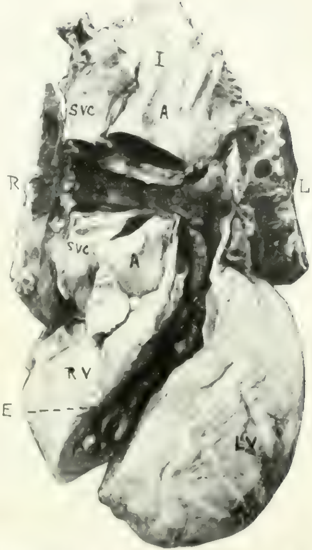

Fig. 5. *Pulmonary embolus in situ. (Letters as in Fig. 4. E. Lower free end of embolus, which still remains in the right ventricle.*

(From the Pathological Museum, University of Sheffield.)

tion was possible, and prostatectomy was arranged for. The man seemed well in every way until two days before operation, when, whilst sitting up for his morning wash in bed, he suddenly fell back, the breathing became irregular, he turned pale, the pulse stopped, and, after a few inspiratory efforts, he died. The total duration of the symptoms was estimated at five minutes, and there was no cyanosis.

A careful examination of the systemic veins failed to show any source of the clot, but the probability is that it arose in the prostatic plexus.

There are many points of interest about this specimen. The pulmonary artery and its branches, for some distance into the lungs, are completely obstructed. The whole of the packing and coiling of the embolus must have taken place during the period, estimated at five minutes, which elapsed between the first symptom of distress and the cessation of the heart.

It would seem as though the heart must have stopped early, perhaps within a few beats after the arrival of the clot in the pulmonary artery, otherwise there would have been some collection of blood on the right side with cyanosis. This sudden cessation of the heart-beat is supported by the falling back, pallor, and cessation of radial pulse noted. Meanwhile it is evident that local thrombosis of the blood passing into the pulmonary artery with the embolus must have been rapidly produced, for it can be seen that, in addition to the coiled and twisted embolus, there are large thrombi forming moulds or casts of the vessels, which must have been manufactured *in situ.*

In this case, therefore, we may assume that death was due to syncope produced by the sudden advent of the embolus.

It should be noted in this specimen that the embolus, in spite of its length, has completely passed through the pulmonary orifice, none remains in the ventricle.

In the next specimen (Fig. 5) for which I am indebted to the kindness of Mr. Archibald Cuff, the patient had been successfully operated upon by him at the Sheffield Royal Infirmary, for some bladder trouble two weeks previously. He was doing well, and seemed convalescent. There had been no suspicion of any vascular thrombosis. One morning early, he sat up in bed, and suddenly fell back dead.

There was no cyanosis.

In this case, the whole of the clot had not got through the pulmonary orifice, and one can form some opinion as to its size and length from the long end dangling down into the right ventricle. This being so, the folding or plaiting is less marked than in the previous specimen. Death was even more sudden than in the previous case, and was undoubtedly due to syncope, the heart must have stopped immediately the front end of the embolus reached the pulmonary artery, so that there were no more systoles to drive on the rest of it. In this case also one sees the local thrombi alongside the embolus.

To complete my remarks on pulmonary embolism, I now refer to the third specimen, less striking in appearance than the others, but of great interest. As the autopsy was made in somewhat unusual circumstances in a private house, it was impossible to adopt the measures required to obtain a perfect specimen.

For the specimen, and the history of it, I am indebted to Mr. W. Birch Caley, by whom the case has been recorded (*Quarterly Medical Journal*, Vol. XI. 1903). The patient, a man, æt. 55, had a simple fracture of the right tibia and fibula about two weeks before. He went on well without any symptoms of thrombosis, or anything of an untoward character, until about 4 o'clock one morning, when, on turning in bed, he was seized with intense thoracic pain and dyspnœa. He lived for half-an-hour, gradually becoming more cyanosed, and fully conscious to the last. The pulse continued after respiration had ceased.

In this case, death was obviously not due to syncope, but to gradual asphyxia, in the common sense of that term. The embolus was not nearly so long, it did not quite fill the artery. As the heart went on beating, there was no chance for local thrombosis to occur. The first entry of the embolus into the pulmonary artery did not cause syncope, but it did cause intense thoracic pain. This fact is of great interest. It may be that the intensity of the pain in the other two cases was so great as to completely inhibit the heart immediately; or it may be that there was no pain in the other cases, because there was no distension of the right heart, such as must have been acute in this.

There are certain features common to these three cases.

In each of them death was quite unexpected, there was nothing to call attention to a thrombus being in the process of formation. In each of them, the embolism occurred just after the night's rest, on slight movement, not getting up, merely sitting up in bed. A great length of clot must, therefore, have been but lightly adherent to the walls of the vein ; probably that length of clot, or most of it, had been recently formed, possibly during the preceding night.

ADDENDUM.

In any autopsy on a case of sudden death, the question of its being due to a pulmonary embolus must arise. If the ordinary procedure of removing the thoracic organs is adopted, it may be somewhat difficult to settle the point satisfactorily owing to the disturbance of the pulmonary artery and its branches in the process. In such cases, I always teach students to look for a pulmonary embolus immediately after opening the pericardium. This is done by making a small incision with scissors or scalpel along the conus arteriosus in the direction of, and up into, the pulmonary artery. If a pulmonary embolism has caused death, it will be readily seen through this opening, and a further dissection *in situ* may be made by dividing the aorta and slitting up the pulmonary arteries. If, however, it is desired to preserve the specimen, the opening should be closed and the whole of the thoracic viscera removed *en masse*, starting at the trachea, and turning them out from behind. They should then be placed in a formalin solution of some kind, and, after thorough hardening has occurred, the lungs may be cut off except a small piece at each root. The aorta is then cut away where it lies over the right pulmonary artery, and the front wall of the pulmonary arteries can be laid open showing the embolus *in situ* (as in Figs. 5 and 6).

THE ANTISEPTIC ACTION OF METALLIC SUTURES.

By CHARLES LEEDHAM-GREEN, M.B., F.R.C.S.,
Surgeon to the Queen's and Children's Hospitals, Birmingham; Assistant Lecturer in Bacteriology, Birmingham University.

[With Plates VIII. and IX.]

SOME little time ago, whilst experimenting upon the relative value of certain antiseptics, my attention was drawn to the evident power, which some of the pure metals possess, of inhibiting the growth of micro-organisms. That certain of the metals have this property is well known to bacteriologists, but, so far as I am aware, the subject has received but little attention at the hands of the surgeon. I, therefore, venture to describe briefly some experiments, bearing on this point, which have, I think, a distinct clinical importance.

A number of small wire rings was made of the various metals which are used, or are capable of being used, in surgery. After being sterilised, by boiling or heating in the open flame, and then allowed to cool, these rings were severally dropped into Petri dishes containing liquefied gelatine or agar medium, which had recently been inoculated with the staphylococcus aureus. After the medium had solidified, the plates were incubated, the gelatine ones at the ordinary room temperature, and the agar ones at 37° Cent. From day to day the plates were examined and their appearance noted. In the case of the majority of the metals tested, the presence of the wire loop in the gelatine seemed to have little or no influence on the growth of the micro-organism. The colonies of the staphylococcus grew freely and evenly throughout the medium, and as readily about the wire ring as at a distance from it. On the other hand, it was at once evident that some of the metals had a distinct and powerful inhibitory action upon the growth of the micro-organisms. In these cases the culture-medium in the immediate neighbourhood of the wire loop remained free from bacterial growth, thus forming a clear, transparent halo about the metal ring, the degree of inhibitory, or bactericidal, power being indicated by the width of this halo. A microscopical examination of the plates entirely confirmed and emphasised what was apparent to the naked eye. The width of the

PLATE VIII

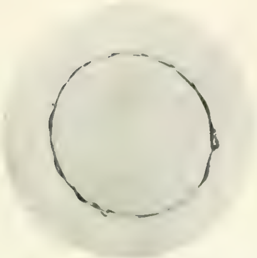

Fig. 1. Silver wire in gelatine culture of staphylococcus, showing no halo of inhibited bacterial growth.

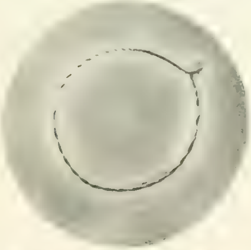

Fig. 2. Silver wire in gelatine culture of staphylococcus, showing faint halo of inhibited bacterial growth. N.B.—A weak culture was used and grown slowly at a low temperature.

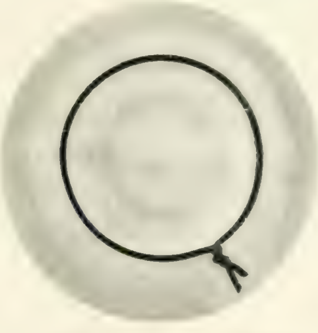

Fig. 3. *Brass wire in gelatine culture of staphylococcus, showing a wide halo of inhibited bacterial growth.*

Plate IX.

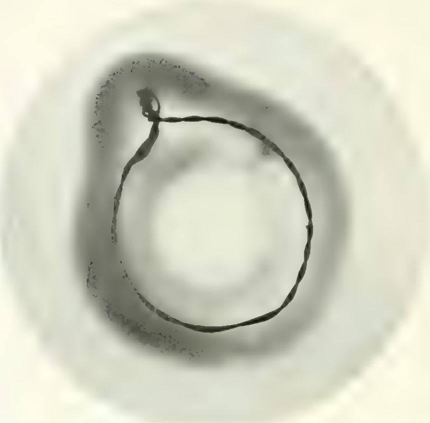

Fig. 4. *Bronze wire in gelatine culture of staphylococcus, showing a wide halo of inhibited bacterial growth.*

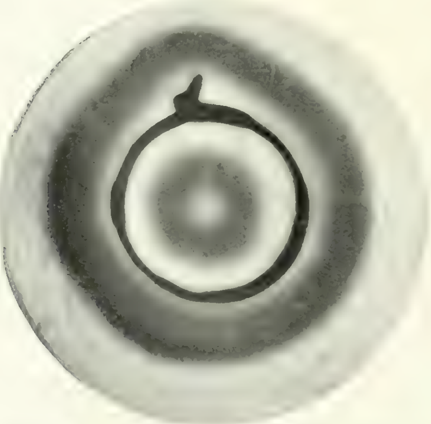

Fig. 5. *Copper wire in gelatine culture of staphylococcus, showing a particularly wide halo of inhibited bacterial growth.*

inhibited area varied according to the rapidity of the growth of the micro-organism. The agar plates, which were incubated at 37° Cent., showed generally a narrower zone than the gelatine ones grown at the room temperature. In the same way, when a micro-organism of rapid growth, such as bacillus proteus, was substituted for the staphylococcus, the massive growth frequently spread over the plate without any regard to the presence of any of the metals. On the other hand, when steps were taken purposely to delay the development of the culture, as by incubating at a low temperature, or by inoculating the medium with a weak and sensitive microbe, not only was the halo more marked in the case of those metals, which showed it under ordinary conditions, but a narrow inhibited zone could be seen surrounding metals which did not usually exhibit this property. Indeed, by the aid of other and more delicate experiments, to be described later, it could be demonstrated that all the metals possess some inhibitory power, even such apparently insoluble ones as gold and platinum, though, for all practical purposes, this degree of inhibition seems far too slight to be of surgical importance.

The result of this experiment showed that the following metals possess practically no inhibitory power :—Gold, platinum, tin, aluminium, magnesium, nickel (Fig. 1). The same remark applies also to lead and silver, save that when weak cultures, grown at a low temperature, were used, they showed a slight inhibitory action (Fig. 2). Iron exhibited a variable action, dependent on whether oxidation was favoured or hindered. If no oxidation took place, then no inhibitory action could be detected ; when, on the other hand, there was free oxidation, the inhibitory action was decided.

By far the most powerful of the metals, bactericidally, proved to be copper, and, in a less degree (according to the percentage of copper used in their composition), its alloys, bronze and brass (Figs. 3, 4, and 5).

Similar experiments were made with screws and nails of copper, bronze, brass, steel, and silver-plated steel. The result was the same as previously, copper and bronze proving decidedly superior to the others. The silver-plated screws used were those advocated by Mr. Arbuthnot Lane for the treatment of fractures. The silver-plating was, however, so thin that the steel oxidised almost as freely as in the unplated screws, and had an equal inhibitory power. When the screw had been specially thickly plated with silver, then oxidation was prevented, but the inhibitory power vanished.

An interesting variation of the experiment was to infect the various metal loops, or screws, with a watery emulsion of the staphylococcus, and, after drying the metals, to carefully place them on the surface of a sterile agar, or gelatine plate. The result showed that, whereas in the case of the metals with little or no inhibitory action, a powerful growth of the staphylococcus appeared on the medium, in that of the copper and bronze frequently no growth took place, or, if it did, it was but scanty.

In selecting a metal for use, as either a temporary or a permanent suture material, many qualities are requisite quite apart from any question as to its inherent antiseptic virtue. But, provided that the necessary qualities of ductility, tenacity, and freedom from irritating properties can be found in a metal, which also possesses a distinct antiseptic action, it is, I think, obvious that such a metal would be the most suitable, and that, other things being equal, the stronger the antiseptic power the more suitable the metal is as a suture, so long as it is unirritating to the tissues.

Practically, only two metals are used in England for this purpose, viz., silver and iron or steel ; the former in the form of wire, the latter occasionally as wire, but principally as screws or nails for bone-suture. It is somewhat strange that silver should be used so extensively for this purpose, for, apart from its non-irritating quality, it has little to recommend it, for not only is it costly, but its tenacity and ductility leave much to be desired, and, as we have seen, its antiseptic power is insignificant.

From the latter point of view, iron and steel are superior, though only when oxidation takes place, and this quality is purchased at the expense of some irritation to the tissues. On the Continent, bronze wire has largely supplanted silver as a suture material, where it is used in the form of aluminium-bronze (copper 95 parts, aluminium 5). This wire far surpasses silver in tenacity, pliability, and, as we have seen, in its antiseptic properties, and has proved itself clinically to be quite unirritating to the tissues. It is, however, too soft a metal to permit of its use in the form of screws or nails for bone-suture. For this purpose, I have lately been employing a true bronze (*i.e.*, a mixture of copper and tin), which has the requisite hardness, does not irritate the tissues, and is equally powerful as a permanent antiseptic agent.

I have been especially struck with the value of permanent antiseptic sutures, such as bronze, in the fixing together of bone fragments, where the wound is already infected, *e.g.*, in certain compound fractures. Such sutures are far better tolerated, and are less likely to lead to troublesome sinuses than sutures possessing no antiseptic value.

Pure copper, though possessing the greatest antiseptic action, is generally regarded as being irritating to the tissues. I have too little clinical experience of it to speak with any certainty on this point, but in those instances in which I have known of its use no sign of irritation was produced.

REMARKS ON THE MEASUREMENT OF THE NECK OF THE FEMUR.

By J. HOGARTH PRINGLE, M.B. (Ed.), F.R.C.S.,

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It has often seemed to me that the value of measuring the length of the neck of the femur, as a clinical aid to the diagnosis of injuries, or of disease with resulting deformity about the hip joint, has hardly been appreciated as it deserves, and that the method is not carried out so often as it might be. Perhaps one reason to account for its apparent neglect is to be found in the inconvenience experienced in carrying it out, for it must be admitted that some of the methods are scarcely practicable for practitioners, who see this class of patient single-handed. At the same time, I fancy that there can be little doubt in the mind of anyone, who has practised it carefully, in a few cases in which there has been a question as to the head or neck of the bone being involved, that information of great value is to be obtained by this investigation.

Nowadays, it is not merely a matter of the diagnosis between a possible intra-capsular fracture of the femur and a contusion of the hip, though this is important enough, and, in the case of greenstick fracture, may be a matter of very considerable difficulty ; but we have now to diagnose cases of coxa vara from disease of the hip joint of all varieties, and occasionally from injury. Moreover, it is often apparently as the result of what ultimately turns out to have been very slight violence, or a strain, that attention is called to the condition of a hip joint which is in a state of coxa vara.

In coxa vara and greenstick fracture of the neck of a femur, which, as Whitman has endeavoured to show, and I believe correctly, is much more common than was once supposed, the difference in the measurement of the two limbs may be quite small, and a greater degree of accuracy is therefore demanded for its determination, as errors in measurement readily creep in. We must measure between fixed points, and these can only be bone landmarks. Now two of these, the anterior superior spine and the great trochanter, are not just so readily, nor so accurately, defined as might be supposed, due mainly to the mobility of the skin and soft tissues over them, and, in the case of the great trochanter, the depth at which it lies from the surface. I find that the anterior superior spine will frequently be most variedly marked in the same patient by several people, who will, in consequence, obtain an equally variable measurement of the line between the anterior spine and the internal or external malleolus, so that, for many years, I have been in the habit of taking as the "anterior superior spine" the angle formed by the insertion of Poupart's ligament into the iliac crest. The ligament is sufficiently rigid to form a distinct and easily palpable angle with the bone, into which a tape measure can be firmly pressed regardless of mobile skin over it.

The methods, for the estimation of the comparative length of the necks of the two femora most commonly employed, are the determination of the relation of the great trochanter to Nélaton's line, and the comparison by Bryant's triangle. As is well known, in the normal individual, the great trochanter should just touch Nélaton's line, a line drawn from the ischial tuberosity to the anterior superior spine, but I find that, in quite a number of limbs which are apparently normal, the tip of the trochanter is well above this line, and, in such an individual, say one who has a slight degree of coxa vara on one side and therefore with one femoral neck just a little shorter than on the other side, it will be necessary, for the purpose of determining the variation in length between the two sides, to measure the actual distance at which the tip of the great trochanter lies above Nélaton's line on each side, and this, to any one single-handed, will frequently prove to be a distinct difficulty.

The method of Mr. Bryant by the employment of his well-known "triangle" is often a most useful one, and especially in the gross injuries, such as a complete fracture of the neck of the femur; but it is often troublesome to work, and it has so frequently happened, in my own experience, that several people will get such entirely different measurements by this method that, although I formerly constantly employed it, I seldom do so now, unless for purposes of demonstration. I find that, in the case of patients who have only a very slight difference in length between the two femoral necks, it is not easy to accurately estimate it by this method on account of the difficulty in getting the vertical line (the patient being supine) of the triangle accurately perpendicular, and, in cases of slight coxa vara or partial (incomplete) fracture of the neck, any inclination of the vertical line will produce an error in the measurement of the horizontal line, the one by which the shortening of the neck is estimated. In his original description of his method, Mr. Bryant wrote that these two lines of the triangle could be marked out by placing one finger vertically at the anterior superior spine, and a finger of the other hand at right angles to it, opposite the great trochanter. In a gross lesion, this may be quite sufficient, but it is by no means accurate enough for the slighter degrees of shortening. There is one point of information to be obtained by the Bryant triangle not given by any other method, which is sometimes useful in cases of coxa vara, and that is the comparison of the "oblique" line of the triangle, *i.e.*, the distance between the anterior superior spine and the great trochanter, for, as was, I think, first pointed out by Professor Ogston, this varies with, and, therefore, gives the amount of rotation of the limb.

A method, possibly less known than these two, but one that is often of service, is that of Professor Chiene, who recommended that two lines should be taken, one between the two anterior superior spines, and the second between the two great trochanters. With the neck of each femur normal, these two lines should be parallel, while in the case of shortening of the neck of one femur the lower line inclines to, and makes an angle with, the former. But in the slighter degrees of shortening, I find that to get an approximately accurate idea, it requires two people who can recognise the bony points, and the second person is not always available. There is a further disadvantage in having to estimate, usually to guess at, the angle of divergence, which may lead to inaccuracy later on in the course of the case, when one desires to decide whether the condition is improving or progressing.

Then there is the method recommended by Mr. Morris, the estimation of the distance at which the outer aspect of the greater trochanter is placed from the symphysis pubis.

It is generally stated, regarding this measurement, that, in fracture of the neck of a femur, the distance is shorter on the side of the injury. I have often employed this method for comparison with the others, and have found that it is altogether uncertain in the results obtained. Probably, in the majority of cases, the distance is shorter on the injured side, but I have very often found it the same length as, and frequently greater than, that of the sound side. By itself, I do not consider that it is in any way trustworthy, and it has the very distinct disadvantage of requiring a special apparatus.

In my own practice, I have constantly employed all of these methods, but, for reasons indicated, have not always been satisfied with them, and, for some years past now, have adopted a method, which is at once simple and easy to carry out, and which, after a considerable experience of it, seems to me to be thoroughly trustworthy. The method is not mentioned in any book published in English, dealing with fractures etc. about the hip joint, with which I am acquainted, and I had never been able to find any indication that it had ever been suggested, until quite recently, when I found that Hoffa, in the 4th edition of his book on fractures, mentions it as having been proposed by Wight, but he gives no reference, and I have not been able to discover the original paper.

It is preferable to measure it with a regular measuring tape, so that a definite idea, in inches or centimetres, of the difference between the two sides can be obtained, but it can be quite efficiently and easily carried out with a piece of string or tape. In that case I measure from the tip of the great trochanter to the tip of the external malleolus, in order to determine, in the first place, if this portion of the limb is of the same length on each side, or it may be measured from the great trochanter to the most prominent point of the external condyle (really the ridge of the groove for the poplitæus tendon); this will eliminate any inequality in the length of the limb from the knee downwards. With the information that the limbs, or, at any rate, the femora below the trochanters, are of the same length, I measure from the anterior superior spine to the tip of the external malleolus (or poplitæal ridge of condyle) on both sides; if this measurement is less on one side than the other, it signifies shortening of the neck of that femur. Of course, any serious deformity of the shaft of one femur will tend to throw out this measurement, but such cases are very rare.

The following are brief notes of one of the last of the cases, which have been under my care, and which illustrate some of the points referred to in the foregoing.

H. F., aged 25 years, admitted to Glasgow Royal Infirmary, November 1st, 1906, on account of pain in the left hip joint, attributed to a strain three weeks previously. He had been off work and in bed for fourteen days preceding admission, and came to hospital, as it was believed that he had some acute inflammation in the hip joint. On examining the patient, I found that the movements of the right hip joint were perfect and painless, whereas those at the left caused a certain degree of pain, and that, after flexing through 40°, the pelvis began to move with the lumbar vertebræ. The great trochanter on the right side was 2·5 c.m., and on the left side 3·5 c.m. above Nélaton's line. Measurements were made as follows:—From great trochanter to external malleolus: right side, 88 c.m., left side, 88 c.m. From anterior superior spine to external malleolus: right side, 93 c.m., left side, 92 c.m., so that there was one centimetre shortening (barely $\frac{3}{8}$ inch) of the left femoral neck. This, along with other signs present, indicated that the patient had a very slight degree of coxa vara. It will be observed that, by Nélaton's test, the right great trochanter was 2·5 c.m. (1 inch) above the line, which, as I have said already, when one has to deal with small variations, necessitates a direct measurement on both sides, in order to determine how much, if any, shortening may be present. Röntgen photographs by my colleague, Dr. James Riddell, showed, on the right side, a perfectly normally shaped femoral neck, and, on the left side, a very slight curvature of the neck, thus confirming the diagnosis of coxa vara. All the information required, regarding measurement, was obtained by the method here advocated, and my reason for bringing it forward is that it is easy to work, can be carried out single handed, and does not render it necessary either to move a patient, or for the examiner to change his position from one side of the bed to the other. Moreover, it does not appear to be known, or, at any rate, to be made use of in this country.

PHYSICAL METHODS OF TREATING HEART DISEASE.

No. 3.—Diet.

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DIET IN HEART DISEASE.

In every condition of bodily health or infirmity, diet must, of necessity, take rank as a matter of first-rate importance.

The procuration of food, being as it is one of the primal instincts of created beings, alone renders a reference to the subject a matter of more than passing interest.

A recognition of this fact has caused it to enter, not only into every system of medical treatment, but also into the religious and social life of nations. So much is this the case that, in the older civilisations of the East, we find, according to Sir Richard Burton, that certain dishes, whose preparation was a matter of the nicest detail, were served only at the tables of kings, and that special permission was required for subjects to partake thereof even in the privacy of their own homes.

Amongst the ancient Greeks, no system of athletic training could be considered complete, without a proper amount of attention being paid to the question of food and drink, while among the Jews, the observance of fasts and festivals furnishes a good example of the importance attached to it on the score of religion.

At the present day, we find the same condition of things prevailing ; the same critical regard as to the form, in which the various constituents of the body are to be replaced, the same anxiety on the part of athletes or "physical culturists" and their trainers, and mentors to ensure that the proper amount and quality of food is consumed as in the days of ancient Greece.

While diet is of such vast importance to those in good bodily health, it becomes of even greater consequence to those in delicate conditions.

A due appreciation of this is made the dominant note at most health resorts, and I have little hesitation in saying that, on account of a proper regard being paid to its importance, the great majority of patients, deriving benefit from their stay at such places, owe their improvement almost as much to the judicious regulation of their dietary as to the various balneotherapeutic prescriptions.

It is hardly going too far to assert that the general tendency is to eat rather more than is necessary for our physiological requirements.

The possession of a good appetite is often regarded as a matter for congratulation by its possessor, but its complete satisfaction is only too frequently a subject for regretful consideration.

The fact is ignored, or not appreciated, that it is not the amount consumed which is of consequence so much as the amount assimilated.

It is not my hope that, on such a well-worn topic, anything new will proceed from my pen, but I will at all events endeavour to be practical.

With this object in view, I shall, so far as possible, leave out of consideration any of the probable chemical changes, which occur in the various articles of food during or after their absorption. For, when all is said and done, we know remarkably little that can be called really definite with regard to them, while clinically our advice is sought for guidance as to quality and quantity of food rather than its caloric value.

I shall therefore content myself with a discussion of foodstuffs, their quantities and preparation, and the position that some special articles of diet may at times occupy in the scheme of treatment.

For the reasons above advanced, although we may have at times to advocate the adoption of a restricted dietary, and perhaps insist upon the necessity of limiting the food to one particular item, we should never allow ourselves to forget that

> "Man is a carnivorous production,
> And must have meals, at least, one meal a day.
> He cannot live like woodcock upon suction,
> But, like the shark and tiger, must have prey.

The assertion of the fact that the purpose of food is to supply energy to the organism is, according to Kassowitz, a one-sided statement, because it ignores its purpose of serving for the construction of the organism. The growing individual can only grow by means of the substances furnished by food. Accordingly, every organism, including the full grown, can replace, what is lost through physiological function or disease, only by food. For this duty it is not its caloric value which is of clinical interest, but its power to take part in the building up of the body.

In addition, there are various mineral substances which are essential for the growing as well as for the full-grown man.

Before going any further, I should say that there are certain figures, which need to be remembered, whenever the question "What shall I eat?" is propounded. They provide for a mixed dietary for a man doing moderate work and, with a little thought, can be turned to account at a moment's notice. The essential elements of a diet are proteids, fats, and carbohydrates in certain definite proportions, viz.:—

Proteids, 4·5 per cent., represented by ½ lb. uncooked meat.
Carbo-hydrates, 14 per cent., represented by 26 ozs. of bread.
Fats, 3·0 per cent., represented by 2-3 ozs. butter.

The half pound of meat may, for practical purposes, be represented by the lean of two good chops.

Twenty-six ounces of bread is equal to about half a loaf.

A two-ounce pat of butter forms a cube about 1¼ inch.

With these quantities and their several bulks always present to one's mind, it is a comparatively simple matter to estimate with fair accuracy whether a stated quantity of food is or is not sufficient in any given case.

Without some such mental picture, it is almost impossible to form a correct notion of what amount of food is being consumed by the very ambiguous statements of the patient or his attendants.

When fish is the main constituent of the meal, the amount is more readily ascertained, because so little allowance has to be made for the presence of bone. Generally speaking, its food (proteid) value may be looked upon as identical with that of meat.

In heart disease, the impairment of the circulation of the stomach renders it necessary that all foods partaken of should be of the easiest digestibility, and of the highest nutritive value. For the same reason, it is desirable to select food that will not place an undue amount of work upon the liver. More, inasmuch as the liver decomposes uric acid, the great cause of high arterial tension, it is important to see that there is no *excess* of purin-containing food ingested. Since it is hopeless to expect to be able to eliminate purin-containing bodies totally from the circulation, seeing that they are produced by the cells of the living tissues, I feel that it is best to free one's mind from the idea that meat is an article of diet to be avoided, and to recognise the fact that, after all, it presents the readiest means of introducing into the system the requisite amount of proteid material.

Moreover, it may be rightly urged, in dealing with cases of heart disease, that a preference for solid food should be exhibited, so long as possible, because it occupies less space in proportion to its nourishing properties, while its presence in the stomach encourages peristaltic action, and thus favours circulation.

A method of preparing meat, which I have frequently employed, is that recommended by Dr. Salisbury. It is simple, but nevertheless requires the greatest care in its preparation.

Take half a pound of the best part of the round of beef, cut thick, free it from all fat and shreds of fibrous tissue. Place on a gridiron, or, if one is not available, in a frying pan with a piece of butter about the size of a hazel nut. If a gridiron is used, it should be heated, and the bars rubbed with suet, to prevent the meat being branded. Broil or fry over a very good fire for five minutes, turning once. It should not be turned with a fork, but with a pair of tongs, or a large flat spoon. This will prevent the juices escaping. It should next be passed three times through a mincing machine. I think the best type of machine to use is one, in which the meat passes through perforations, against which the blades lie. This machine not only minces the meat very thoroughly, but any fibrous tissue left behind becomes entangled either in the perforations or around the blades. When this occurs to any extent at the outlet, the meat is returned through the inlet. The blades must then be cleaned. I am entering into these practical details, because I have found the difficulty of making even intelligent people understand the importance of preparing the meat in the proper manner. Moreover, in giving instructions, it is as well to let the nurse, or whomever the responsible person may be, understand that there is a working acquaintance with the methods advocated. Life after all is made up of details, and success depends, in a large measure, upon the amount of attention bestowed upon them.

As the meat emerges from the machine it should be received with all its juices into a thoroughly clean basin. It may then be stood upon the range, covered over with a plate or saucer, and the temperature maintained at about 130° F., a point well below the coagulation point of serum albumen, which I presume may, in this respect, be regarded as a typical proteid. The coagulation of serum albumen commences at 163° F., and is complete at 185° F.

The meat should then be served as required on a hot-water plate, without either salt or pepper. If it is not kept hot up to the moment of being eaten, it is very unpleasant, and no invalid will partake of it. It takes 2–3 hours for the digestion of 200 gms., say, six or seven ounces.

The difficulties of preparation are so obvious that it is unwise to leave it altogether in the hands of an ordinary domestic servant.

For one thing, the temperature will not be properly observed. A touch on the outside of the basin will be considered quite sufficiently accurate to enable the "experienced cook" to decide whether it is likely to be done or not, as if the meat were placed there for the purpose of finishing off the cooking process. Nothing is more misleading than the attempt to estimate the temperature by means of the hand. I have, over and over again, tested the vaunted powers in this respect of bath attendants, and have found that even experienced men and women make errors of upwards of 30 and 40 degrees. The use of a thermometer is essential.

Another reason for paying so much attention to the temperature of the meat is because the analysis of meat, as an article of food, is usually that of its raw condition. Meat, under the conditions here described, is being employed as a re-constituent, and not merely as a savoury preparation to tempt an unwilling appetite. If, then, the analysis is to be of practical value as a guide, there must be no risk of altering it by cookery. I do not lose sight of the fact that the process of cooking may actually enhance its nutritive value, but this is certainly not the case if it has been overdone. A plateful of cremated meat is not the substance the analysis of which we are turning to account.

Again, it must be remembered that the methods of preparation recommended obviate the use of the oven. Of all pernicious inventions for dealing with meat, the close oven of the ordinary ranges undoubtedly carries off the palm. I greatly prefer the oven of the gas cooking stove, inasmuch as in it there is a nearer approach to the old-fashioned method of roasting. The bottom of the oven is open, and the gas-jets are situated around it. The perforations in the roof allow the empyreumatic vapours from the cooking meat to find their way up the flue, and so out into the chimney. The oven can be heated to such a degree, before the meat is placed in it, that the albuminous coating of the meat is coagulated very rapidly after its introduction. There is no loss of nutrient material, any fluids which do escape are caught in the dripping-pan over which the stove stands.

The methods of preparing fish are by broiling, frying, and boiling. The first two methods are to be preferred. In frying it is important that the oil or fat, in which the fish is placed, is very hot, not too scanty in amount, and that the fish should be freed from all superfluous grease before being served.

To prevent charring by the gridiron, it is a good plan to rub the bars with chalk. In boiling a certain amount of the nutritive substances escape into the water.

Other proteid-containing foods are cheese, peas, beans, and lentils. According to analysis, these all contain a higher percentage of proteid material, and are of greater caloric value than meat. However, comparatively few people care to subsist entirely upon them, and they are not to be commended as foods for invalids with delicate digestions because of their very imperfect absorption. Dr. Salisbury in his book, *Food in Health and Disease*, draws particular attention to this point, and incidentally emphasises the fact, which is generally recognised, both by the laity and the medical profession, that man, in the process of his evolution, has determined that a mixed diet is that which enables him best to meet the exigencies of his struggle for existence and supremacy. If there were any truth in the saying, "Dis-moi ce que tu manges, je te dirai ce que tu es," man would prefer to remain in the commanding position he has attained in the scheme of creation, rather than chance his future degradation to the vegetable kingdom!

While this may be regarded as the general feeling of the majority, one must in fairness admit that a certain number find a vegetarian diet most suitable to their special idiosyncrasies, and that it is possible to construct a completely purin-free diet, which will supply tissue waste, and be of equal caloric value to meat. Its chief drawbacks lie in the lack of variety, the difficulty of digestion, and the far greater harm which would result from imperfect cooking than would be the case with meat. A more serious objection is that there is said to be some loss of vigour, and a lowering of the power of resistance to the onset of disease.

With regard to the digestibility of such a diet, it has been estimated that over 40 per cent. of the proteids of legumens is lost on account of the inability of the intestines to absorb them, and that a plateful of peas, beans, or lentils takes from four to five hours to leave the stomach. The best method of preparing them for food is to mash them.

An attempt to subsist upon green vegetables is foredoomed to failure. The rôle that fat plays in diet needs no special reference beyond the general statement that in cases, in which there is a peculiar tendency to fat formation, and in fatty degeneration or more commonly infiltration of the heart, the fats ingested should be limited. In these circumstances, it may indeed be necessary to eliminate fats as far as possible from the diet, especially in cases, in which the stomach is dilated and its motile power impaired, in which there is also a tendency to heartburn and flatulence, or in which an icteric tinge gives rise to the suspicion that the liver is not acting as efficiently as it should.

Having thus briefly considered the proteids and fats, the next essentials to engage attention are the carbohydrate materials.

The main sources of their supply are bread, rice, tapioca, arrowroot, potatoes, and sugar, in short, all starch-containing bodies. They may be looked upon as of the first importance in the production of muscular energy, but this fact must not make us forget that the proteid bodies are also capable of sustaining a similar rôle. Carbohydrates undergo both very perfect digestion and absorption but not in the stomach so much as in the small intestine.

The great objection to their use, in cases of lowered vitality, is their readiness to undergo fermentation in the stomach and intestines. A fully distended stomach, pressing upon a flabby, weak heart, may by its pressure occasion syncope or even death. It is therefore a matter of practical importance to diminish the supply of carbohydrate material. In doing this, one may as well bear in mind the following points: —

1. The liability to promote fermentation.

2. As a general rule, a "heart patient" is not fed with the object of providing material for muscular exertion.

3. In such cases, meat and other proteid materials will sufficiently replace the carbohydrates for the purpose mentioned above, and more efficiently as tissue builders. There is a difference between muscular energy and muscle tissue, which it may be of value occasionally to remember.

Thorough cooking is essential for rendering starch-containing foods digestible. Boiling in water causes the starch granules to swell, and burst their containing envelopes. Baking converts the starch granules into dextrine, which is more easily digested than starch, because it is soluble, whereas starch is insoluble in water. A potato is more readily made digestible by boiling, bread by toasting.

Having now discussed the several constituents of food as they are found in their commonest solid forms, it may be as well to consider the methods by which they may be administered in their liquid forms.

The two commonest and readiest means, by which the several elements of food may be thus introduced into the system, are by soups and milk. If the patient is not able to eat solid food, any soup given him should be made from fresh meat as well as bones. It is not sufficient to make soup from bones alone, as such soup contains practically no nutritive value. They may, however, be boiled in water, which has been utilised for the purpose of boiling meat, and thus a fairly nutritious soup is obtained. It will not, however, make such good soup as water in which meat has been boiled, with the definite intention of preparing soup therefrom. When this is the case, the fresh meat is placed, or at any rate ought to be placed, in cold water, which should be slowly brought to the boil. It is a good plan to add a few drops of hydrochloric acid to the water.

A very excellent method of making soup or beef-tea is as follows :—

Crush up a few bones, and place them with half a pound of beef steak in a large gallipot, which should contain about a pint of water in addition. This should be stood in a saucepan of water which may be slowly raised to boiling point. A few drops of hydrochloric acid will dissolve out some of the bone salts as well as hasten the dissolution of the albumen. During the cooking process, long before boiling point has been reached, all the removable albumen of the meat will have been dissolved. For reasons already advanced, it is better not to run the risk of elevating the temperature in the gallipot above a certain degree, so, as soon as the water in the outer saucepan boils, it should be removed from the fire, and stood in a warm corner of the range. It should then be covered with a plate to prevent the access of soot or smoke, but not so completely as to prevent evaporation.

The whole apparatus can be left in that situation until the desired degree of concentration has been reached. A large amount of nourishment can be given in this manner in a very small space, and thus one of the objections to fluid food removed. With this method of preparation, it is quite unnecessary to employ tinned meats and extracts. I do not know what its food value is, but it has always seemed to me better when the HCl is added than it is without.

It is not at all an uncommon thing for heart patients to suffer from indigestion, often of a painful character. This may lead to aerophagia. Flatus accumulates in the stomach, the heart has the additional burden of a distended stomach to contend against. Pain, partly cardiac, partly gastric, increases till with the eructation of the pent-up gases, relief is suddenly obtained. When this occurs, it is often difficult to prevent a patient becoming persuaded in his own mind that the chief cause of his trouble is gastric, and that if only it could be relieved, his cardiac condition will improve also. Another symptom, which I have known to be complained of, and which I believe to be due to the gastric disturbance, is a great longing for something to drink.

It requires some amount of assurance to inform such a patient, who may make this symptom one of his chief causes for complaint, that he must endeavour to limit the amount of liquid, which he consumes, to 2 or perhaps 2½ pints a day. I have had very few patients, who have been able to bring themselves to this pitch of self-denial, but, on the other hand, thirst is not invariably a prominent symptom.

The last patient I met, who was making it a serious complaint, came to me at the latter end of last February in order to undergo a course of Nauheim baths. The following was his condition. He was only able to walk at a very slow pace, any excitement would cause palpitation and dyspnœa, which were either accompanied, or followed, by anginal pains, which he referred to the left side of the chest. Pulse small, weak, irregular, vessel hard, no œdema. Heart impulse neither visible nor palpable. Heart sounds irregular, varying in intensity, neither sound could be described as clear. The area over which they could be best heard was from an inch and a half outside the V.N.L. to nearly the mid-axillary line. As regards his build, his bones gave one the impression of being those of a slight man, but, when he was put on the weighing machine, I found he weighed 13 st. 9½ lbs.

In addition to the baths, I felt it necessary to remodel his dietary very thoroughly, and, amongst other instructions, he was recommended not to exceed 2½ pints of liquid in the twenty-four hours. He carried out the latter part of the prescription with truly heroic fortitude. His weight declined and his general condition improved while under treatment. He was out of my care practically all the summer, and returned, a month or so ago, looking very much better and slighter. He had adhered rigidly to the diet which had been ordered, his thirst was much less troublesome, stomach less dilated, cardiac impulse both visible and palpable, though still very irregular, and his weight has fallen to slightly under 11 st. He underwent another course of baths and exercises, and is, by the latest account, "walking 8 miles daily and feeling very confident." He never exceeded the 2½ pints of liquid during the whole of the hot summer weather.

I make special mention of this case because, in my first article (November, 1900), I laid stress on the fact, that, in my opinion, the Nauheim baths and Schott exercises were not to be regarded as the be-all and end-all of the spa treatment of heart disease, but that baths, exercises, rest, diet, and drugs should all be pressed into the service as occasion demanded.

Milk is the most important of all foods, because it contains the elements of a diet in such proportions that life may be supported on it alone for indefinite periods. It is only slightly deficient in carbohydrate material, but this defect can be so very easily remedied by the addition of sugar, or by utilising a portion of the daily allowance for the purpose of making arrowroot, that it can hardly be regarded as a matter of moment.

The quantity of milk necessary to yield the requisite amount of proteid is five pints. It is particularly well adapted for use amongst invalids, as it is so readily digested, is unirritating, and is said to diminish the tendency to fermentation, when used alone. It can be readily prepared and administered, and fills the dual rôle of food and drink.

In spite of all these advantages, there are, notwithstanding certain drawbacks, which always have to be borne in mind. It is a bulky food, inasmuch as nearly 90 per cent. is water. This alone may be sufficient to condemn it for use in some cases of heart disease, or under some conditions of treatment of aneurysm, and in cases in which there is much dropsy. The desirability of milk-feeding, in cases of aneurysm, must always be considered, and by no means be lightly dismissed. It has a certain tendency to aid coagulation, a point to be remembered in cases in which there is marked inclination to thrombosis and venous stasis.

Patients occasionally complain of constipation when on a milk diet, but this in itself is a slight matter, and is due partly to the fact that it is an unirritating form of food, and that it undergoes such very complete absorption. Another complaint is that it is apt to cause headache. I have frequently met patients who have refused milk entirely on this account. Other objections which I have noted are a sense of lassitude, especially if the milk has been taken in the middle of the day, while, if taken the last thing at night, there is, invariably in some cases, a peculiarly disagreeable taste in the mouth on first waking in the morning. I have tried all manner of expedients to obviate this most disagreeable effect, but have not found anything really completely satisfactory. The best is to wash out the mouth with a solution of Acetozone, 1 per 2,000.

It is a very largely prevalent idea among the laity that, inasmuch as milk is a liquid, it is unnecessary to do more than drink it. But though people, in normal health, can consume it in bulk, with more or less impunity, this is not the case with invalids. The quantity of milk ordered for the day should be given at stated intervals, and in definite quantities. It should be sipped slowly, and not gulped down like an unpleasant draught which had to be speedily disposed of. If this is not done, curds may form in the stomach, which may completely defy the action of the gastric secretions, and pass on into the small intestine as a solid mass, which may travel the whole length of the alimentary canal without undergoing any material alteration.

Before the stomach has had time to get rid of the first mass, another lot of milk may arrive in the same manner, and give rise to a considerable degree of discomfort. The patient will then complain that he cannot digest milk. I have seen very large masses of curdled milk vomited up, which have simply been formed as the result of such hasty consumption.

Confinement to bed is not an essential condition for the satisfactory employment of a milk diet. I have repeatedly allowed patients, restricted to this form of nourishment, to take moderate exercise. It is only when the quantity of milk is to be restricted that confinement to bed may have to be insisted upon.

The methods of preparation of milk in the form of junket, and curds and whey, or by peptonising, are so well known that it is unnecessary to do more than indicate the necessity or remembering that such methods of administration vary the monotony of the diet in a very agreeable manner.

There is, however, a preparation of milk, which has hardly received the attention which its virtues merit, I mean kephyr, or, as it is more ordinarily called, "koumiss." I believe that there is in reality a difference between koumiss and kephyr. Koumiss is the term applied to fermented mare's milk, while kephyr is the fermented milk of the cow or goat. I was at one time under the belief that koumiss was the term applied to the unfermented mare's milk, and, as I used to partake of it daily, I formed a very poor opinion both of its taste and its sustaining properties. It is neither so sweet nor so rich as cow's milk, and altogether forms a very poor substitute for it. It is very much better in its fermented condition, and has a pleasant, slightly acid flavour. There are two ways of preparing kephyr. The first is rather a troublesome proceeding, but as I consider it rather superior to the second, I will describe it in detail. If mare's milk is used, the amount of water must be diminished as well as the proportion of the sour milk or added koumiss.

Dilute a quart of milk with about one-fourth its quantity of water; to it add about a quarter of a pint of very sour milk, or old koumiss, or kephyr. Stir thoroughly, cover with a cloth, and place in an earthenware or wooden basin in front of a fire. Leave it for twenty-four hours undisturbed. At the end of that time remove the cloth, and stir briskly with a whisk, thoroughly breaking up the coagulum, which will be seen on the surface of the milk. Re-cover it, and allow it to stand for another twenty-four hours, at the expiration of which period it may be again whisked. Now it is New Kephyr. It should be bottled in soda-water bottles, and the corks wired down. Fermentation continues after bottling. It may be used at the end of another day, but it improves in quality up to the third day, when it is at its best, and generally very strongly effervescent. For this reason it should never be kept in a warm place, unless the weather is very cold. When opening a bottle it is as well to take the precaution of wrapping each bottle round with a napkin in case it should burst, and pointing the bottle away from any person who may happen to be in the room, as the cork is often driven out with considerable force. Some of the New Kephyr should be kept for the purpose of making a second brew, which is generally of better quality than the first.

The second method of preparation is more rapid. Take a quart of new milk, add to it a tablespoonful of moist sugar, and the same amount of brewer's yeast. If this is unobtainable, German yeast will do. Allow fermentation to proceed for a few hours, and then bottle as before.

Koumiss is the only spirituous liquor obtained from animal sources. In addition to the alcohol and carbonic acid, it retains all the essential properties of the original milk. There is no doubt that the trouble of preparing it constitutes the great bar to its use; otherwise it forms a palatable food-beverage, of considerable nutritive value, readily digested, and of good stimulating power.

Eggs naturally claim attention next. I fear that the statement I am about to make will sound almost heretical, but it is my belief that, unless an egg is properly cooked, it forms the most indigestible kind of food one can be called upon to eat. I think that, for an invalid with a feeble circulation, such as one is at the moment contemplating, a fried egg is absolutely impermissible. Poaching an egg is taking a step in the direction of over cookery, but eating a fried egg is courting disaster. The albumen of the egg is very close to, in fact, practically in contact with, the bottom of the pan, and is frequently burnt. The more heat to which an egg is exposed, the firmer and tougher becomes the albumen, until it more nearly resembles leather than any other substance with which I am acquainted. A boiled egg should never be eaten, if boiled long enough to become hard. If the patient cannot eat rather underdone eggs he had better do without them altogether. An uncooked egg is very much more digestible than a cooked egg, though perhaps not so palatable. If objection is taken to them on this account they may be administered in the form of an egg flip, or, as what I have heard described, as a "prairie oyster." For the benefit of those who may not have come across eggs treated in this way, I may explain that a prairie oyster is prepared in the following manner:—An egg is broken into a wineglass, a few drops of vinegar added, with a very little pepper and salt. The whole can be swallowed without difficulty, and tastes something like an oyster. I have known patients to get quite fond of them.

Tea and coffee should never be taken with meat or eggs. A tannate of albumen may be formed, which will prove quite indigestible unless the tea or coffee has been very carefully made.

Apart from this, these beverages are on the whole suitable in cases of heart disease, especially where a stimulating effect is desired. In addition to being stimulants, they possess certain diuretic properties, which may also be of advantage, but, as this effect is associated with a rise of blood pressure, it should make one cautious about ordering or permitting the use of strong tea. I have, in a certain number of cases of insomnia, noticed that an improvement in sleeping has followed the use of tea and coffee. Such sleeplessness has in all probability been due to some want of vascular tone. A point in the history of such cases has been that they are often able to sleep after dinner, so long as they are in a more or less upright position, *e.g.*, sitting in a chair, but as soon as they get into bed all desire to sleep has left them. I think, in the majority of such cases, that to prescribe tea or coffee is rather like recommending a hair of the dog that bit, because such a form of insomnia not infrequently results from arterial degeneration, or loss of tone, consequent upon persistent high tension. According to Haig, such high tension is very liable to follow tea drinking. Consequently, although relief may be obtained by its exhibition, I am not at all clear that it is a wise prescription, even though it may meet with the patient's cordial approval.

With regard to the question of alcoholic beverages, there are obviously times when, although one may recognise the part they have played in producing certain conditions, a prohibition of their continued use is like bolting the stable door after the steed has been stolen. On the other hand, one frequently meets cases, in which a word of warning, with regard to the unwisdom of their employment, may be followed by very good results. It not infrequently happens that the habitually temperate man is the warmest in resenting advice to discontinue their use. He has never in his life drunk to excess, and he quite fails to realise that such advice is given with a view to the future, and to the possible postponement of the evil day. He can only see in such advice either a warped judgment, or a reflection upon himself, and fails to recognise that there are many people, who have a peculiar tendency to high arterial tension, who may be most moderate eaters and drinkers, and yet a plate of meat, a cup of tea, a single glass of champagne may result in headache, insomnia, irritability of temper, and even gouty manifestations. In such cases, vegetarianism, or total abstinence, is not a matter of choice but of compulsion. We must recognise the idiosyncrasy, but it is another matter altogether to argue therefrom, that all people must be strict vegetarians, teetotallers, or cocoa drinkers, as the case may be. A broad view must be taken of the individual as of the race. He should not be forced, perhaps unnecessarily, into the narrow ways of preconceived opinion. The personal equation must never be lost sight of, because there is no such thing as a standard individual. The nearest approach to one is the average man. For him, the rule of life can be summed up in the one word "moderation." Yet what may constitute moderation in the man in the days of his youth and vigour, may be excess in those of his maturity and old age. As he advances in years, building material is not required to the same extent as formerly, nevertheless too frequently no alteration is made in the amount consumed to meet the altered and daily altering conditions of his life.

The integrity of the nervous system, upon which ultimately depends the power of the body to resist the onslaughts of disease, is more likely to be undermined by too high than by too low living. Very few drugs create a demand for their continued supply on the part of the nervous system so rapidly or insistently as does alcohol. For this reason its excessive use is so prone to be followed by ill-health.

When it is ordered medicinally, the conditions under which it is to be taken, together with the amount, ought to be prescribed just as every other drug is. The patient should not be left to exercise his or her own discretion, for too frequently a habit may be acquired which may cause untold misery. On the other hand, an effort suddenly to transform an elderly patient, who has all his life been in the habit of taking stimulants, into a total abstainer is a proceeding singularly destitute of wisdom. Some people may accomplish the feat, others may be rendered both miserable and ill by such a complete departure from long-established custom.

If a patient with heart disease is able to get along comfortably without alcohol, it is obviously much better that he should do so. The nervous system has already quite enough to do to meet the legitimate demands of the heart, struggling to accomplish its task against heavy odds, without the evanescent and altogether artificial stimulus of alcohol. It is very seldom that good is gained by applying whip and spur to an over-tired but willing horse. Keep the alcohol, if possible, as an emergency drug.

The only remaining constituents of food, which are left for consideration, are the salts. The commonest of these, chloride of soda, is normally present in all the tissues of the body, but especially in the body fluids. Owing to this fact, the therapeutic value of salt has been rendered somewhat difficult to estimate. From Zambelli's observations, it seems evident that so long as salt is being ingested, in cases of œdema of cardiopathic origin, cardiac tonics have little effect, until diuresis has been promoted by the exhibition of diuretics and a salt-free diet established.

Bearing in mind the fact that salt water is capable of acting as a very free cathartic, it seems at first sight somewhat remarkable that its presence in food should so hinder the elimination of dropsical fluids. The reason may possibly be that, when taken in sufficient quantity to act as an aperient, the glands of the stomach and intestine may be stimulated to such a degree by the salt solution as it passes along, that absorption of the salt does not take place to any appreciable extent, while that which is absorbed is re-excreted, and thoroughly washed out by the secretion from the irritated glands, thus producing the purging effect. When, on the other hand, it is taken in the small amounts usual when taken with food, it is quite readily absorbed, the quantity of salt in the blood is increased, and, as it normally has a greater affinity for the fluids rather than the cells of the tissues, it transudes into the connective tissue spaces, renders the œdematous fluid more saline, and thus actually promotes further exudation into the already saturated tissues. From this it would appear rational to employ active saline purgatives in dropsical conditions.

The chief value of green vegetables as articles of diet lies in the presence of their mineral constituents.

DISEASES OF THE STOMACH.

A REVIEW OF SOME RECENT WORK.

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METHODS OF DIAGNOSIS.

On the basis of A. Schmidt's assertion that raw connective tissue is digested by gastric juice, but is unaffected by pancreatic juice, Sahli,[1] early in 1905, introduced a new and, according to him, trustworthy method for determining the functional capacity of the stomach, which did not involve the use of the stomach tube. The principle of the method consists in the administration of substances such as iodoform, potassium iodide, methylene blue, salol, which following absorption are demonstrable in the secretions, enclosed in gutta-percha bags secured by raw catgut, which is digestible by the gastric juice only, and noting the time of appearance of the particular substance employed in the secretions. Such bags, designated "desmoid capsules" usually containing iodoform 1½ grain, or methylene blue ⅓ grain, or both, in pill form, are administered immediately after the chief mid-day meal with a draught of water. The urine passed 5 hours and 7 hours later, and again next morning, is examined for the presence of blue discolouration, or by the appearance of such after boiling with concentrated acetic acid for the presence of chromogens, where methylene blue has been employed. The appearance of the substance in the secretions, within seven to twelve hours, indicates a normal functional capacity. Earlier, it indicates a condition of hyperacidity, whilst, further delayed, it points generally to subacidity, or, if a test breakfast has shown the existence of free acid, to a condition of hypermotility.

Kaliski[2] investigated the value of Sahli's method in 24 cases having different degrees of acidity, and virtually confirmed the conclusions arrived at by the originator. For the construction of the capsules, he gives the following noteworthy details, small squares, × 4 cm. of gutta-percha tissue are cut ; on the centre of each three or four pills are placed, and then the corners are taken up, twisted to exclude as much air as possible, and firmly secured by three turns of raw cat-gut 00. previously soaked in cold water for a minute, and twice knotted on the same side. Before use, the capsules are placed in water to prove they are watertight and that they will not float.

Einhorn[3] tested the reaction, in four cases of achylia gastrica, and in all he obtained positive results, from which he concludes that the method is useless, and is based on an erroneous assertion.

Alexander and Schlesinger[4] found in a number of normal and pathological cases, the greatest irregularities in the appearance of the reaction. In normal cases, with constant acidity the reaction appeared either irregularly, or not at all. In one particularly striking example, they found a positive reaction in the urine, 2½ hours after the administration of methylene blue, whilst iodine was demonstrable in the saliva not until 20 hours after the administration of potassium iodide. Similar irregularities were noted in cases of hyperacidity, whilst in cases of anacidity, and atony, a positive reaction was obtained. It would thus appear that the desmoid reaction presents so many inexplicable irregularities, as render it useless for practical purposes.

Boas,[5] in an interesting article, maintains that the test-breakfast properly applied gives the least fallacious results. It is important to adhere rigidly to the following directions :—The breakfast should be given to a fasting patient on an empty stomach. The presence of stagnating stomach contents vitiates the results, accordingly, these should be previously removed, and the stomach washed out ; the breakfast should consist of white bread, 35 grammes, and water, or simple infusion of tea, without any addition, 400 ccm. ; it should be removed from the stomach exactly at the end of one hour. Beyond determining the hydrochloric acid in the removed test-breakfast, it is important to estimate the peptic and milk-coagulating powers of the mixture. The presence of blood, bile, mucus, saliva, and stagnant material in the stomach may render any estimation of hydrochloric acid entirely fallacious. In the absence of hydrochloric acid, conclusions as to diminished motor power of the stomach can only be justified by a quantity of stomach contents of from 150 to 200 grammes. Thus it may happen that a test-breakfast reveals achylia, and gives a residue of 120 grammes without any gastric manifestations whatever.

Lastly, it is to be noted that various nervous disturbances, menstruation, and pregnancy are liable to cause transitory variations in the acid secretion from anacidity to hyperacidity. Another interesting source of error has been demonstrated by Bartenstein,[6] who found that different portions of the stomach contents often gave different acid values, the portion last removed generally being the most strongly acid.

For the accurate determination of the chemically active hydrochloric acid in the gastric contents, the hydrolytic method of Hoffmann has been utilised by Whitney,[7] who suggests the following somewhat complicated procedure :—

Taking four well-stoppered flasks, he places in

1. 10 ccm. of filtered stomach contents + 2 ccm. of methyl acetate ;
2. 10 ccm. $\frac{N}{40}$ hydrochloric acid + 2 ccm. of methyl acetate ;
3. 10 ccm. of filtered stomach contents ;
4. 10 ccm. of distilled water + 2 ccm. of methyl acetate.

The flasks are incubated for four hours at a temperature of from 50°—55° C., and then the contents of each are titrated with decinormal soda, phenolphthalein being used as indicator. The results obtained in the individual flasks then are in terms of $\frac{N}{10}$ NaOH.

1. The amount of free and combined HCl, + the amount of acetic acid liberated from the methyl acetate by the hydrolytic action of active HCl present.

2. The amount of HCl in 10 ccm. $\frac{N}{40}$ HCl, + the amount of acetic acid liberated by the HCl.

3. The amount of free and combined HCl, originally present in the stomach contents, ± the effects of incubation.

4. The amount of acetic acid liberated by the hydrolytic action of distilled water.

From these results there can be determined :—

A. The amount of acetic acid liberated from the methyl acetate by the active HCl in the stomach contents, in terms of $\frac{N}{10}$ NaOH = 1 − (3 + 4).

B. The amount of acetic acid liberated from the methyl acetate by the active HCl, in 10 ccm. $\frac{N}{40}$ HCl in terms of $\frac{N}{10}$ NaOH =

2 − (4 + value in $\frac{N}{10}$ NaOH of 10 ccm. $\frac{N}{40}$ HCl) ;

and, these obtained, it follows that

B : A : : percentage of HCl in $\frac{N}{40}$ HCl : percentage active HCl in stomach contents.

Moore, B.,[8] applies the same principle, somewhat differently and with less complexity, calculating the amount of active hydrochloric acid present from the *rate*, at which hydrolysis of the ester takes place.

Willcox[9] determines the physiologically active HCl volumetrically as follows :—1. 20 ccm. of filtered stomach contents are diluted by the addition of 40 ccm. of distilled water ; to this 10 ccm. of pure nitric acid are added, and about 5 ccm. of a solution of iron alum. The total chlorine is then determined by precipitation with excess (30 ccm.) of $\frac{N}{10}$ silver nitrate solution, and the excess titrated back with $\frac{N}{10}$ ammonium sulphocyanide solution. 2. 20 ccm. of filtered stomach contents are evaporated, on a water bath, to dryness. The residue is incinerated in order to drive off all volatile chlorine, and being taken up in 60 ccm. of water, the remaining chlorine is determined as in 1. The difference between 1 and 2 being the physiologically active HCl.

For the detection of lactic acid in the stomach contents, Croner and Cronheim[10] recommend the following method capable of revealing ·0025 per cent. :—Two grams of potassium iodide are dissolved in 5 ccm. of water, and to this is added one gram of sublimed iodine ; the solution is filtered through asbestos or glass-wool, and water added to 50 ccm. To this solution five ccm. of aniline are added, and the mixture stored in a dark stoppered bottle. For use the bottle is shaken, and to a few ccm. sufficient 10 per cent. caustic potash solution is added to make it strongly alkaline; the addition of filtered and boiled stomach contents produces, if lactic acid is present, either spontaneously or on warming, the characteristic odour of iso-nitril.

For the determination of the ferments, pepsin and rennin, or better, the proteolytic and milk-coagulating powers of the stomach contents, Illoway[11] describes the following methods:—Pepsin.—In 10 ccm. of stomach contents, removed one hour after a test breakfast, he places 10 grams of coagulated egg albumen, and incubates the mixture at 38° C. Where the secretion is normal, the albumen is entirely dissolved in 5½ hours. In cases of hyperacidity the time for complete solution may be less, signifying the presence of excess of pepsin. Excess of acid alone does not accelerate solution of the albumen, in fact, it often happens that digestion is more rapid with small acidity than with great. He distinguishes a condition of hyperpepsinia, where digestion occurs in from 3 to 4 hours; of normal pepsinia and of hypopepsinia, where the time exceeds 5½ hours.

Rennin.—Illoway uses an extension of Leo's method, inasmuch as he always makes two experiments. To each of two portions of milk, 10 ccm. and 20 ccm. respectively, he adds 5 drops of gastric contents. Under normal conditions, coagulation occurs in the 10 ccm. in from 4 to 15 minutes, in the 20 ccm. in from 15 to 30 minutes.

He finds that rennin may be present in normal amount where the pepsin is deficient, and *vice versâ*, and further that, where pepsin is entirely absent, rennin, which is the last to disappear, may still be present in the gastric juice.

The importance of the microscopical examination of the contents of the fasting stomach is emphasised in an interesting paper by Ackermann and Gompertz.[12] The finding of unaltered epithelial cells and leucocytes denotes the absence of active gastric juice; of cell nuclei alone indicates its presence. The source of any mucus is denoted by the kind of cells with which it is associated. Squamous cells point to œsophageal, cylindrical cells to gastric origin. The finding of blood and pus in the absence of signs of stagnation, and with a diminished or absent

HCl secretion, suggests cancer of the lesser curvature situate between the cardia and pylorus. In the stagnation, caused by a recent cancer originating in an old ulcer, it is noteworthy that the gradual disappearance of HCl from the gastric juice is associated with the disappearance of sarcinæ and the appearance of long bacilli.

FUNCTIONAL DISORDERS.

The relation of the acidity values, as determined in the stomach contents after a test-breakfast, to the manifestations of disordered gastric functions, is significantly dealt with by Kaufmann.[13] He finds that the limits, within which the values of the total acidity and the free HCl may vary without manifestations, are, for the total acidity, 5 to 90, for the free HCl, 0 per cent. to ·2 per cent. Accordingly, it would appear that, where analysis gives values not exceeding these upper limits, or where the secretion of the HCl is absent, that any manifestations, which may be present, cannot be attributed alone to the chemical composition of the gastric juice. Even in cases, which give values higher than these limits, it is questionable whether such degrees of acidity can alone produce manifestations. On the other hand, it is equally certain that such manifestations as pain, acid eructations, waterbrash, heartburn, are commonly associated with high acidity values, in particular with high values for free HCl, which thus appears to be a condition for their origin. In such cases, however, the acidity values rarely reach the upper limits met with in normal individuals, and they very exceptionally exceed them.

It follows, therefore, that the symptoms of so-called hyperchlorhydria must depend, not merely on the degree of acidity of the stomach contents, but much more on some concomitant factor. A view also rendered probable by the results of dietetic treatment, which, whilst ameliorating the symptoms, may in no way influence the acidity values, and further by the fact that the introduction of large quantities of hydrochloric acid into the healthy stomach fails to produce manifestations of hyperchlorhydria.

Similar are the relations which obtain between symptoms and subacidity.

Inquiring for the concomitant factor, or factors, answerable for the subjective manifestations, Kaufmann obviously regards the organic lesion, as in cases of subacidity, secondary to catarrh, or cancer, or of hyperacidity with ulcer. Where, however, such organic lesions are absent, he seeks the factor, in the first place, in disturbance of gastric motility. Dealing with 53 cases, he found a pathological increase in size of the inflated stomach in 35, and, as the remaining 18 were typical neurasthenics, he seeks the basis of the symptoms in an excessive susceptibility of the gastric mucosa. The latter class improve, under general measures directed towards the nervous basis of the condition, the former under measures which improve the motor power of the stomach.

Disturbance of the secretory functions of the stomach very frequently obtains with disease of the female genitalia.

In 41 gynæcological cases in which the patients complained of gastric trouble, Winkler[14] examined the secretory and motor functions of the stomach, and found HCl normal in 11, diminished in 20, absent in 5, and excessive in 1. In 17, the total acidity was normal, in 2, it was excessive. The secretory functions were normal in 29·9 per cent., in spite of the manifestations. In 18 cases, he found a condition of gastroptosis, associated in the majority with motor insufficiency. From the therapeutical standpoint, it is of interest that he found that treatment of the gynæcological condition had practically no effect on the gastric disturbances.

In 22 cases of melancholia of varying degrees, Cowie and Inch[15] found, in the majority, a condition of hyperchlorhydria of moderate degree, together with increased peptic powers, and increased motility, results which confirm the previous observations of v. Noorden and other writers.

Relating to the secretory disorders of the stomach, a number of interesting observations, specially having regard to treatment, have been published. Hyperchlorhydria and hypersecretion are clearly differentiated by Riegel,[16] the former, as a qualitative anomaly of secretion, characterised by the production of excessive quantities of hydrochloric acid in response to the taking of food, the latter, as a quantitative anomaly, characterised by the production of excessive quantities of gastric juice, either in response to the stimulus of food (alimentary form), or continuously apart from the taking of food. Hypersecretion is frequently, but not invariably, associated with hyperchlorhydria. The view that hypersecretion depends on deficient motor power of the stomach, permitting the passive accumulation of secretion in that viscus, he strongly controverts, believing any dilatation, which may obtain, to be, as a rule, secondary, and due to spasm of the pylorus induced by a protective reflex from the duodenum. Nor can gastric ulcer be regarded as the origin of this anomaly of secretion. Deficient motility of the stomach may, in some cases, be a predisposing factor, but that it is not invariably primary is indicated by failure of operative measures to remedy the condition. Clinically, hyperchlorhydria and alimentary hypersecretion are alike in their manifestations. Where hypersecretion becomes complicated with motor insufficiency and dilatation, the dominant manifestations may be those of the latter condition, but, even then, certain important features are noticeable, differentiating such cases from those of primary dilatation. There is the excessive secretion of gastric juice in response to the stimulus of food, and in the alimentary form in response to that stimulus alone. The proportion of fluid in the extracted stomach contents is greater than in simple dilatation, the separation between the fluid and solid constituents is more distinct, and the specific gravity of the fluid is less. Again, whilst in simple dilatation the difference between the total acidity and the free hydrochloric acid is relatively great, in hypersecretion it is small, owing to the relatively small amount of HCl which becomes "bound." With regard to treatment, he recommends, in hypersecretion, complete rest for the stomach for a time, nutrition being maintained per rectum, and then a diet of albumen and fat; of drugs he advocates atropin, as it diminishes the secretion of gastric juice, and lavage in the evening.

Albu[17] recognises four types of hyperchlorhydria—(1) a true secretory neurosis—a frequent manifestation of gastric neurasthenia; (2) chlorotic; (3) pre-ulcerous; (4) gastritic—in chronic hyperplastic gastritis. In treatment, dietetic measures hold the premier position. The diet should consist chiefly of albumin and fat. Lean meats, fresh butter, milk, olive oil, and eggs.

Fat meats and fish are prohibited. Vegetables require careful selection; no raw or uncooked vegetables, and only such cooked vegetables, as are finely divided, and freed from pieces of cellulose and husks, are to be allowed. Stale white bread, Zwieback, toast, and biscuits are well borne.

Small quantities should be taken at each meal.

In order to influence the secretion of hydrochloric acid, he recommends one to two teaspoonfuls of olive oil, to be taken from one to two hours after each meal. Of drugs, he advocates belladonna, and prescribes the following:—

Extr. Belladonnæ, gr. 5.
Bismuth subnit. }
Magnes. ust. } āā gr. 150.
Sod. bicarb. }

Of which a dose of 30 grains is to be taken one to two hours after food. He also mentions Bergmann's "Magenkau-tabletten," which contain an alkali and pilocarpine, as proving beneficial. Hypersecretion he divides into six groups:—

(1) A complication of gastrectasis, the most common form; (2) a complication of slight disorders of motility-atonic; (3) a complication of gastric ulcer; (4) a complication of hyperchlorhydria; (5) an independent form, the continuous hypersecretion of Reichmann; (6) the intermittent form, belonging to the series of central neuroses.

The divergence of opinion, as expressed in this classification, from the views of Riegel are very striking, and, bearing in mind the results of Kaufmann's investigations [*v.s.*], would indicate that our knowledge of these functional disorders of the stomach is still very superficial.

During recent years, the administration of fat in the form of olive oil, or almond oil, has been advocated by a number of observers, in all conditions in which there is secretion of hydrochloric acid in excessive quantities—hyperchlorhydria, hypersecretion and gastric ulcer—a procedure which is now becoming dignified by the appellation "oil cure." Its advocates claim, and justly so, that the oil: 1. Diminishes the amount of HCl secreted; 2. it is nutritive; 3. it is soothing, diminishing thereby any tendency to pyloric spasm, and in, if an open ulcer exists, forming a protective salve to its base. Cohnheim, one of its more enthusiastic advocates, recommends the use of olive oil, in doses of from 1 to 2 ounces three times a day, from half to one hour before each meal. He has seen no untoward effects, and in cases of pyloric stenosis, spastic or organic, hyperacidity, etc., its exhibition has been of great utility.

Merkel's[15] experience also is favourable, in all cases he found the administration of oil to be followed by an increase in body weight and in strength, and he regards its use as specially indicated in irritative spasm of the pylorus, and consequent delayed emptying of the stomach.

Blum,[19] who has given the method a trial in Ewald's clinic, in cases of hyperacidity, with and without hypersecretion, and with and without dilatation, of chronic gastric and duodenal ulcer, and of pyloric stenosis, finds that it is not so well tolerated, as would appear from the writings of Cohnheim. In 18 cases, the oil was well tolerated in five only, in the other 13 it aggravated the discomfort, and had to be stopped. The five cases were hyperchlorhydria and hypersecretion, in which the exhibition of the oil produced a distinct, but temporary diminution in the acidity of the gastric secretion, but this was not permanent. On the other hand, a definite improvement in the nutrition of the patients was noted.

Silver nitrate is a drug, which was formerly more extensively prescribed in hyperacidity and gastric ulcer, and gained a favourable reputation. The action of this salt on gastric secretion has recently been investigated by Baibakoff,[20] and with interesting, if anomalous, results. In a number of cases of gastric disease of various types, he found that silver nitrate, administered in doses of from $\frac{1}{30}$ to $\frac{1}{3}$ grain in pill form, three times a day, or by lavage of the stomach with 500 ccm. of a 1 in a 1,000 solution, increased the amount of hydrochloric acid secreted, the peptic power of the gastric juice, and also the motility of the stomach. Accordingly, he concludes that its use is contraindicated in hyperacidity and ulcer, and is called for in cases in which the secretion and motility are defective.

Under the designation *Gastromyxorrhœa*, Kuttner[21] describes a condition, characterised by an excessive production of mucus in the empty fasting stomach, an amount of mucus exceeding 25 ccm. obtained from the fasting organ, he regards as abnormal. The condition is not uncommon, and clinically may belong to one of two types—the intermittent and the chronic.

In the former, there occur at irregular intervals, attacks of almost uncontrollable vomiting, the vomit consisting of a slimy fluid, alkaline, neutral, or acid in reaction, and occasionally mixed with blood or bile. Preceding the vomiting, which generally comes on suddenly in the morning shortly after awakening, there is headache, nausea, and anorexia. Pain is conspicuously absent. Such attacks may last but a day, and may closely simulate gastric crises ; more often they extend over three to five or even twelve days, and then subside quite suddenly, leaving the patient very prostrate. With the onset, lavage may arrest the attack, otherwise the only course, as nothing given by the mouth is retained, is the subcutaneous injection of morphia, and this is not always efficient. In two such cases, noted by Kuttner, the condition was associated with hydrorrhœa nasalis.

The chronic form often obtains without manifestations, and is most frequently associated with chronic catarrh, with nervous disorders of the stomach leading to an acidity, and also with chronic ulcer.

A curious case, which in several features is reminiscent of the above-described condition, is recorded by Mathieu and Roux,[22] with, however, a different interpretation. A female, aged 35 years, vomited either once, or as many as six times, during the day, from 50 to 80 ccm. of a sanguinolent slimy fluid, the vomiting being preceded by violent pain in the region of the inner end of the seventh left costal cartilage, radiating thence to the left and through to the back, at the level of the tenth dorsal spine. The pain developed especially during the night, and increased in severity until vomiting occurred, when it subsided. Further constant features were bulimia, weakness, and cold sweats. There were no manifestations of dyspepsia notable after the termination of the attacks.

The condition had existed ten years, and remained uninfluenced by six months' treatment including suggestion.

The vomit, according to the writers, consisted of saliva, and of pharyngeal and œsophageal mucus intimately mixed with unclotted blood, probably of intraglandular origin, which had accumulated in the œsophagus, as a result of spasm of the cardia, and had given rise to the retrosternal pain. As expressing this interpretation of their case, they would term the condition *Hæmosialemesis*.

In the uncontrollable vomiting, occurring in cases of abdominal disease, as peritonitis—post-operative or traumatic—intestinal obstruction, etc., Bennett[23] recommends that the patients should be allowed to drink water, cold or warm, whichever they prefer, as such renders the vomit less foul, more and more clear, and finally, to the benefit of the patient, nothing but clean water is regurgitated. He gives four cases, in illustration of what is but a modification of the washing out of the stomach, as recommended in such cases by continental physicians, viz., chronic ileus, irreducible hernia, following operation for appendicitis and abdominal trauma.

The general belief that gastroptosis is associated with delayed emptying of the stomach, has been closely investigated in 10 cases by Loening,[24] and he has shown that, so far from this actually being the trouble, there is in many cases a marked hypermotility. Gastroptosis *per se* causes either no alteration of motility or an increase. On the basis of this result, he recommends, in cases of gastroptosis, that the left lateral posture be assumed after meals in order to retard the exit of the stomach contents into the duodenum.

(*To be continued.*)

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Prize Essay.

THE TREATMENT OF SCIATICA.

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SCIATICA is understood, in this article, to refer to pain in the course of the sciatic nerve, due to a primary localised interstitial neuritis. It may also occur as a secondary affection from pressure, growth, or inflammation in neighbouring structures affecting the nerve in any part of its course, and may be simulated by various conditions, which must be kept in mind while making a careful physical examination of each case.

Ætiology.—A predisposing cause, such as a toxæmia, septic, rheumatic, or gouty, usually exists, but that the disease does not depend upon the blood state is shown by its being, almost always, confined to one nerve, bilateral cases being extremely rare.

The exciting cause may be pressure on the nerve, severe muscular exertion or strain, and it is significant that the disease is much more common in men than in women. Cold may bring on an attack in a predisposed person, though, in these days of open-air methods, one may be pardoned for doubting whether a "draughty watercloset seat" is sufficient to set up sciatic neuritis.

Pathological Anatomy.—The operation for nerve-stretching has given ample opportunity for observing the state of the nerve, which is found reddened and swollen, with marked exudation into the sheath and interstitial connective tissue; hæmorrhages have been found; the nerve-fibres are only secondarily affected.

Symptoms.—The chief symptom is pain, which may commence suddenly, often during some severe exertion, but more frequently the onset is gradual. The pain is felt in the course of the nerve or its branches, and is described as aching, gnawing, or stabbing; at first it may only be complained of on movement, but later it becomes constant, with exacerbations of great intensity, and is aggravated by any movement which tends to stretch the nerve, or by the increased pressure due to coughing, etc. The patient walks on his toes to relieve the tension of the nerve.

There are points where the pain is felt with greatest intensity ; where the nerve winds round a bone, at the sciatic notch, round the head of the fibula, or malleolus ; where it is exposed to movement, as behind the knee-joint ; or where pressure by muscle occurs, as in the middle of the thigh.

Tenderness on pressure over the nerve is always present, and is an important sign in the diagnosis ; it may render the sitting position unbearable. Alterations in sensation may be found, or complained of by the patient, when cessation of acute pain permits consciousness of them : tingling, pins and needles, and possibly some anæsthesia. Weakness and wasting of some of the muscles supplied by the nerve, or its branches, may be noticed after the disease has been present for some time, especially the calf-muscles and the anterior tibial group ; cramps and fibrillary tremor in the more advanced cases.

These motor and sensory changes are evidence that the nerve-fibres are becoming functionally insufficient from pressure by exudation within the sheath, or from excess of connective tissue formation ; the comparative insignificance of these symptoms in sciatica is due to the fact that the nerve has room to swell. The condition may be contrasted with the same disease in the facial nerve where the bony canal prevents the swelling, and the neuritis causes rapid and complete loss of function, in this case purely motor and so painless.

In sciatica, voluntary movement is rarely lost, nor do the weakened muscles give the typical reaction of degeneration, there is usually slight diminution of response to both faradic and galvanic currents.

Herpes occasionally follows sciatica, and, in rare cases, there is œdema of the leg from vaso-motor disturbance.

There is sometimes an initial rise of temperature.

Diagnosis.—It will be sufficient to enumerate the principal causes of sciatic pain, without discussing the differential diagnosis of each : diseases of the cord and spine, lesions of the cauda equina or spinal cord, chronic syphilitic meningitis, caries or new growth, all of which cause pain, mostly by irritation of the nerve roots. The pain is generally bilateral.

Disease of the sacro-iliac joint.

Pressure by aneurism, or new growths in the pelvis, pressure by the fœtal head.

Referred visceral pain, from inflammatory lesions of the prostate, uterus, or broad ligaments, is not confined to the area of distribution of a peripheral nerve, and there is marked subcutaneous tenderness.

A loaded rectum may determine recurrence, but is hardly capable of originating sciatica.

Diseases of the hip-joint :—Tubercle, osteo-arthritis, coxa vara, and dislocation either simulate sciatica by pain locally and referred to the knee, or set up a secondary perineuritis.

The lightning pains of tabes may, at first, be mistaken for sciatica, but are not often confined to one limb.

Multiple peripheral neuritis affects both sides and there is muscular tenderness.

Foci of inflammation in muscle-tissue.

The pain from a flat foot may be felt severely up the thigh.

It is necessary to enter somewhat more fully into the differences between sciatic neuritis, and neuralgia. "If we recognise that all cases of sciatica, with persistent tenderness of the nerve, are really neuritic, cases of sciatic neuralgia become extremely rare" (Gowers). In neuralgic cases, there is some alteration of the blood-state, such as anæmia, and pain will probably be complained of in other regions ; it is more spontaneous than in neuritic cases and is not influenced by movement, it is often referred to the periphery rather than to the course of the nerve, finally "all cases of definite persistence and prolonged course are inflammatory" (Gowers).

Treatment.—The intractable nature and prolonged course of some cases of sciatica make it necessary to adopt energetic treatment from the commencement.

Rest.—Having determined that the pain is due to sciatic neuritis, the first thing to do is to secure absolute, mechanical and functional, rest for the nerve, and we should warn the patient that the duration of the case will depend, in large measure, on the thoroughness with which this is obtained, and on the persistence with which it is maintained. It is chiefly by want of rest in the early stages that the duration of the disease is prolonged.

Confinement to bed is the best method of securing rest, and whatever influence cold has in exciting an attack, warmth is essential to the treatment. In the more severe cases, it is well to further limit mobility by a long Liston's splint, or a splint jointed at hip and knee so as to enable the position of the limb to be changed, once or twice a day, without disturbing the bandages.

Medicinal and Dietetic Treatment.—Cases, in which there is a rise of temperature at the onset, may be given a saline aperient, and rheumatic or gouty tendencies should be treated by suitable therapeutic and dietetic measures; generally a liberal diet should be encouraged, alcohol, in any form, is probably harmful.

Blue pill will often be found beneficial, partly from its effect in lowering the blood-pressure, and partly from its influence in promoting the absorption of inflammatory exudation.

Counter-irritation should be used early in the treatment; either mustard or iodine may be applied, or a series of small blisters along the course of the nerve at the points of greatest intensity, which may vary in position from day to day, and should be followed by the counter-irritant; the cautery may be used in the same way.

Acupuncture is a method of counter-irritation that may be tried in an obstinate case, before more extensive surgical treatment is determined to be necessary. The method is thus described in Allbutt's *System:* "About six needles are required, these should be, at least, 2½ inches in length, the line of the nerve is first marked in ink or with a dermatographic pencil; then the needles, after being rendered thoroughly aseptic, should be dipped into carbolic oil (1 in 20) and rapidly inserted for 2 inches (dependent, of course, on the amount of fat and muscle to be penetrated), along the course of the nerve.

"Generally the highest needle is about the level of the buttock, and the lowest several inches above the popliteal space. If the operation be skilfully performed the patient only perceives the first needle, and possibly the second, and has no conception that six needles have been pushed into his leg. The needles should be left in position for an hour."

Surgical Treatment.—Old standing cases, which do not yield to counter-irritation, may require surgical interference; the nerve may be stretched, to some extent, by extending the leg and flexing the thigh on the abdomen, but, more efficiently, by an open incision, enough tension being applied to the nerve by the finger to raise the limb from the table. Any adhesions may be freed or the nerve-sheath split longitudinally.

Electricity.—The galvanic current, locally applied, has a marked sedative effect, and will often relieve the pain, but, if this cannot be administered by the practitioner himself, I would strongly advise him not to prescribe the treatment to be undertaken by a nurse, as, to be effectual, large currents must be used which are capable of electrolytic effects, and, if cauterization results, where anodyne effects are looked for, the patient will probably hold the prescriber responsible, and a jury would not accept the plea that counter-irritation, so produced, was beneficial to the patient.

Small currents are useless, on account of the deep position of the nerve in the upper part of its course; 30, 40 or 50 milliampères may be required, and large electrodes must be used, which should be well soaked to reduce the resistance of the skin. With the patient in the prone position, the indifferent electrode is pressed firmly to the corresponding iliac fossa, and the other moved over the tender area; if pain is caused by the current, a larger electrode must be used, or less current passed. The current must be turned on and off gradually, or painful and harmful muscular contractions will result. Temporary alleviation almost always follows, and permanent relief in about half the cases. The treatment should be continued until pain is no longer felt during rest, each application should last from 10 to 20 minutes.

Lewis Jones advises the use of sinusoidal currents in bipolar foot-baths, after the acute stage has passed, the effect on the pain being watched.

The effluve from a high-frequency resonator will sometimes relieve pain, for a time; it has a sedative effect, though, perhaps, is more generally useful as a counter-irritant. A further useful effect of the current is in lowering the general arterial blood-pressure, by dilatation of cutaneous vessels.

A word of warning may be given about its use in cases in which there is latent tubercular focus in the lung, as it causes a marked auto-inoculation with serious, if transitory, symptoms.

In this, as in any other painful affection, X-rays may be used therapeutically; they have a definite effect on nerve tissue, and are more likely to be successful in relieving pain in the superficial part of the nerve course.

Massage.—In the early stages, while the nerve is still tender, massage should on no account be used; but it is of great use later in breaking down adhesions, which are the principal cause of persistence and recurrence of pain.

Baths.—Hot baths are very soothing, relieving pain by the local heat, and by dilatation of cutaneous vessels.

Vapour baths and radiant heat are also useful.

Anodynes.—Where intense pain, or want of sleep, calls urgently for some temporary relief, a hypodermic injection of hyoscin (gr. $\frac{1}{50}$), should be given, which may be repeated daily for months without ill-effects; the use of morphia is not justifiable in cases, such as sciatica, in which the need for an anodyne may be prolonged; cocaine has been proposed as a substitute, but the cocaine habit is quite as demoralising as the morphia habit.

In cases, in which the galvanic current is being used, resource may be taken of its power of introducing drugs locally by cataphoresis; a carbon electrode being used for the positive pole, which is covered with lint soaked in a 10 per cent. solution of cocaine in guaiacol, a current of 5 to 10 m.a. is passed, and complete local anæsthesia is produced in about five minutes; this method of administering cocaine seems the least likely to induce the habit.

(The writer wishes to acknowledge his indebtedness to Gowers's *Diseases of the Nervous System*, Allbutt's *System of Medicine*, Osler's *Principles and Practice of Medicine*, Lewis Jones' *Medical Electricity*, and Hedley's *Therapeutic Electricity*.)

SOME FAMOUS QUACKS.

V.

SIR WILLIAM READ.

OF the methods of quacks in the early part of the eighteenth century, we get a glimpse in a paper by Steele, which appeared in the *Spectator* of July 30, 1712. He begins with a reflection on the general subject which is still applicable to-day, and which will doubtless continue to hold good as long as "this foolish-compounded clay man" continues to exist on the earth. "There is," he says, "hardly a man in the world, one would think, so ignorant as not to know that the ordinary quack doctors who publish their great abilities in little brown billets, distributed to all that pass by, are, to a man, imposters and murderers; yet such is the credulity of the vulgar, and the impudence of these professors, that the affair still goes on, and new promises of what was never done before are made every day. What aggravates the jest is, that even this promise has been made as long as the memory of man can trace it, yet nothing performed, and yet still prevails."

Steele relates how, as he was passing along that day, a paper given into his hand by a fellow without a nose told him what good news had come to town, to wit, that there was now a certain cure for the French disease by a gentleman just returned from his travels. The touch about the nasal deficiency of the quack's emissary possibly needs explanation at the present day, when syphilis is comparatively mild. The destruction of the nose was so common in former days that it was regarded as the sign-manual of the disease. The literature of the Restoration is full of allusions to this characteristic lesion. The paper put into Mr. Spectator's hand set forth that "in Russel Court, over against the Cannon-ball, at the Surgeon's Arms in Drury Lane, is lately come from his travels, a surgeon who hath practised surgery and physic, both by sea and land, these 24 years. He (by the blessing) cures the yellow jaundice, green sickness, scurvy, dropsy, surfeits, long sea voyages, campaigns, and women's miscarriages, lying-in, etc., as some people that has been lame these 30 years can testify; in short, he cures all diseases incidental to men, women, and children."

Our modern quacks, if not more modest in their pretensions, are rather more subtle. They do not, as far as we know, profess to cure long sea voyages or campaigns, and they would probably not call people that had been lame for 30 years as witnesses of their success in dealing with miscarriages and lying-in. But their pretensions are not a whit less absurd.

Steele remarks that "there is something unaccountably taking among the vulgar in those who come from a long way off." We still see the influence of this superstition in the attraction that American quacks, "German specialists," and Indian oculists have for the multitude. Steele points out that "ignorant people of quality, as many there are of such, doat excessively that way"; this, too, might be written of the same class to-day.

The eighteenth-century quack often founded his pretensions on a personal misadventure, which, it is implied, led him to turn his attention to the speciality which he practised. Thus Steele tells us there was a doctor in Mouse Alley, near Wapping, who set up for curing cataracts upon the credit of having, as his bill set forth, lost an eye in the Emperor's service. "His patients come in upon this, and he shews the muster roll, which confirms that he was in his Imperial Majesty's troops, and he puts out their eyes with great success."

In the reign of Queen Anne the eye quack flourished exceedingly. Doubtless this was largely due to the darkness in which the pathology of the eye and the treatment of its diseases were enshrouded at the time. Cheselden, we know, gave some attention to the subject, but it seems generally to have been left to quacks. The Queen herself entrusted the care of her weak eyes to two quacks whom she had sworn in as her oculists in ordinary. One of these was Roger Grant, originally a cobbler by trade, and an Anabaptist preacher. He was a past master in the manufacture of testimonials. His plan, says Jeaffreson, was to get hold of some poor person of imperfect vision, and, after treating him with medicines and half-crowns for six weeks, induce him to sign a testimonial to the effect that he had been born stone blind, and had never enjoyed any visual power whatever till Providence led him to good Dr. Grant, who had cured him in little more than a month. "This certificate the clergyman and churchwardens of the parish in which the patient had been known to wander about the streets in a state of mendicancy, were asked to attest, and if they proved impregnable to the cunning representations of the importunate suitors, and declined to give evidence of their handwriting, either on the ground that they had reason to question the fact of the original blindness, or because they were not thoroughly acquainted with the particulars of the case, Dr. Grant did not scruple to sign their names himself or by the hands of his agents."

The other "sworn oculist" was William Read, one of the most successful quacks of whom there is record. The fact that he was grossly ignorant—being, it is said, unable even to read—only makes his success in gaining fame, wealth, and a title, all the more extraordinary. Dogberry says that reading and writing "come by nature," and many people, who are in a conventional sense "educated," have the same notion in regard to the treatment of disease. Queen Anne's simple faith in her illiterate oculists was like that of "Society" at the present day in bonesetters.

He began life as a tailor, abandoning that calling to become a mountebank, and finally developing into an itinerant quack. His early years are wrapped in obscurity, even the date of his birth being unknown; but from 1687 to 1694 he seems to have wandered through Northamptonshire, Yorkshire, Oxford, Devonshire, Wiltshire, Somerset, Bath, and Windsor, boasting everywhere of his wonderful cures. In 1694, he settled in York Buildings in the Strand, and it was whilst he lived there that he issued the first of a series of blatant advertisements, headed with the motto "Post nubila Phœbus: nihil absque Deo."

The following is a specimen:—"Oculist and Chirurgical Operator, who after 22 years' travel and practice, hath Acquired the true Method of performing all Curable Distempers, Incident to y^e Eyes, as Couching of Cataracts Glaucomas, Suffusions, as his frequent Performances on

Hundreds, in England, Scotland and Ireland may Sufficiently Demonstrate, as well in the Universitye and City of Oxford, upon Severall Blind people, after many years continuance under such Distempers, and who are now (By God's Blessing) restored to their perfect sight that continue after their Couching, which is a Performance that many pretend to, and few perform like him; he does these things upon all Persons before he takes any Money, which is a Confirmation that he is not one of those many Circumforaneous Pretenders that Infest this Land. He also preserves you with approved Remedies y^{t} preserve and strengthen y^{e} Sight in Young or Old, and Infallibly cures paines in the Head, that very often occasions Gutta Serenas, Black Cataracts, that are never cured, but means may be used in time, that may prevent those Obstructions in ye Optick nerves, that occasion them, You may have faithfull Advice of him whither you are Curable or not, and may be Spoken with at his Lodgings from 8 in the Morning till 6 in the Evening.

"Hee cureth ye Poor of Blindness, Cancers, Wenes, Heair lipes, Wry Neckes and Dephness for Charity."

From the last sentence it would appear that Read had as comprehensive a conception of his speciality as if he had known the modern doctrine of the manifold evils that may be caused by eyestrain.

Read is mentioned satirically by Addison in the *Spectator* (No. 547) along with Roger Grant and John Moore, the apothecary vendor of a worm powder, which was said in his advertisements to have brought away worms of incredible length.

Read was knighted on July 27, 1705, "as a mark of royal favour for his great services, done in curing great numbers of seamen and soldiers of blindness gratis" (*London Gazette*, July 30, 1705). He advertised that he was ready and willing to confer those benefits as long as the war lasted, not only on such soldiers and sailors as should need them, but also on the Palatines on their immigration. About the same time he was appointed oculist-in-ordinary to Queen Anne.

Read was now at the height of his fame, and during the same year, 1705, a poem, entitled "The Oculist," appeared, celebrating the Queen's discernment in delighting to honour such a man. The author was doubtless some Grub Street poetaster willing to sell his Muse for a consideration. The following lines will give an idea of the style of his production:—

> "Whilst Britain's sovereign scales such worth has weighed,
> And Anne herself her smiling favours paid,
> That sacred hand does your fair chaplet twist,
> Great Reade, her own entitled oculist.
>
> With this fair mark of honour, Sir, assume
> No common trophies from this shining plume:
> Her favours by desert are only shared—
> Her smiles are not her gift, but her reward.
>
> Thus in your new fair plumes of honour drest,
> To hail the Royal Foundress of the feast;
> When the great Anne's warm smiles this favourite raise,
> 'Tis not a royal grace she gives, but pays."

Other versifiers were not so flattering. Thus Gwynnett wrote:—

> "The Queen, like Heaven, shines equally on all,
> Her favours now without distinction fall.
> Great Read, and slender Hannes, both knighted, show
> That none their honours shall to merit owe.
> That Popish doctrine is exploded quite,
> Or Ralph had been no Duke, and Read no Knight,
> That none may *virtue* or their *learning* plead,
> This has no *grace*, and that can hardly *read*."

Read's knighthood is unkindly alluded to in the following epigram, which refers to Grant, who is variously said to have been a tinker or a cobbler, as well as to Read:—

> "Her Majesty, sure, was in a surprise,
> Or else was very short sighted;
> When a tinker was sworn to look after her eyes,
> And the mountebank Reade was knighted."

In 1706 Read published a book, which he had hired someone to write for him, for, as has been said, he could not even read. It is entitled "A short but exact Account of all the Diseases incidental to the Eyes." The latter portion of this work is occupied with accounts of his cures, and of his invention of "styptic water," which he proposed to substitute in many cases for the barbarous cauterisations then in vogue.

Read's great wealth, and his position at Court, enabled him to mix with the best literary society of his day. Swift, writing to Stella, on April 11, 1711, says of him:—"Henley would fain engage me to go with Steele, Rowe, etc., to an invitation at Sir William Read's; surely you have heard of him; he has been a mountebank, and is the Queen's oculist. He makes admirable punch, and treats you in golden vessels." It is curious to think of a quack oculist entertaining the wits of Queen Anne. The golden vessels make one think of Sir Gorgius Midas.

Read died at Rochester, on May 24, 1715. He was buried in the cemetery of St. Nicholas in that city. His widow, Lady Read, "obedient to the heavenly will," kept on the business in Durham Yard, in the Strand. A mezzo-tint portrait of the oculist, by W. Faithorne, is reproduced in Caulfield's *Portraits of Remarkable Persons*. Another was engraved by M. Burghers.

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NOTES FROM FOREIGN JOURNALS.

TREATMENT OF MELANCHOLIA.

The *Journal des Praticiens* makes the following extract from Masselon's *Étude médicale et psychologique*, which has recently been published in Paris.

The patient must, as a primary condition, be placed in a situation which is best suited for obtaining a favourable result to the treatment, and the latter must be aimed, as much as possible, at the acute symptoms. Isolation answers the former condition, which may be obtained in or out of an asylum.

Asylum treatment is not always necessary, and may be dispensed with, in the case of slight melancholic depression, without anxiety, without symptoms of stupor, and without frenzied ideas. The patient may be allowed a certain amount of freedom, watch being kept on him from a distance. Asylum-treatment may indeed prove actually prejudicial to this class of case on account of the moral shock it may bring about in an over-sensitive patient. But in every case of melancholia, with anxiety, or with frenzied ideas, or those in which the disease has been established slowly and insidiously without appreciable external cause, an asylum is a necessity. The patient must be sent in as soon as possible, as incessant watching becomes necessary on account of the tendency to suicide, and the impulse to commit this occurs quite without warning. The patient, moreover, is dangerous not only to his own life, but parents have been known to kill their children before killing themselves, in order to save these from the opprobium which they believe will be entailed upon them.

The earlier the patient is placed in an asylum, the greater chance there is that the psychosis will decrease. Unfortunately, there is a great deal to be said about the way in which confinement in our asylums is understood. They are not hospitals, but houses of detention for the patients. There is too much noise in these establishments, the patients experience in them too many painful impressions, the medical officer has not time to occupy himself about them, for they are too numerous. Reforms are necessary; the medical staff must be increased, and also the number of attendants, opportunities for disputes must be suppressed; blocks for treatment in bed and for continuous watching must be erected; and the patients allowed more freedom. In some asylums, one principal medical officer alone has charge of 800 patients and more. Watching in a curative sense cannot be instituted, the utmost that can be done is to keep the patients from attempting suicide.

Solitary confinement and the strait-jacket are too frequently made use of, and ought to be banished from the treatment of melancholia. What is especially necessary is rest in bed. This suits admirably those patients who are exhausted, broken-down, or drunkards. Moreover, rest in bed makes the cerebral circulation more regular, and lessens the visual and auditory sensations, which exaggerate the mental anxiety. Unfortunately, it is not always easy to make patients remain in bed, their anxiety makes them want to escape. The attendants must use persuasion and gentle restraint, but never violence. A few of the narcotics and the use of baths may be able to calm down the resistance of the patients. The rest in bed must not be continuous, the patient should get up for two hours every day, in order to prevent the inconveniences which may arise from continuous rest, chiefly loss of appetite and constipation.

The moral treatment will be particularly aimed at capturing the confidence of the patient, and this will only be secured on the part of both doctor and attendant by gentleness, attention to every complaint, and constant, vigilant solicitude. Medical supervision is especially useful, when the affection begins to lose force, when the patient gets perturbed, and begins to be doubtful about the reality of his unhealthy ideas. Reasoning and demonstrations then have a hold on him, and it is then that visits, by those who are dear to the patient, should be paid. At this same stage of the disease it is necessary to take pains to fatigue the patient by suggesting distractions to him, such as visits, theatres, journeys, and excursions. These will tire him, especially if he carries about with him his gloomy humour and his pre-occupations. Hypnotism--and this cannot be urged too strongly, because of the illusions to which this method of treatment may give rise on the part of the relations—never gives favourable results.

The diet must be carefully superintended, the patient must be well nourished so as to ward off exhaustion, but meat must not be given in the case of those of slow digestion, obstinate constipation, and foul breath. A diet of milk and vegetables is often employed with advantage, and a few stimulants may be ordered, tea or coffee. All food must be well prepared and served up, for the least appearance of negligence will cause the patient to allege the bad taste of the food as a reason for refusing to take it. It is often necessary to exhort him and press him to take his food, to make fresh inducements, and to give liquid food which will be taken more easily. Recourse to the feeding-tube must only be made after systematic refusal to feed himself. It is exceptional treatment, somewhat brutal, and has the disadvantage of exasperating patients. Very few melancholics indeed need this treatment, for, as a rule, by dint of insisting, begging, returning to the charge, and threatening, the physician succeeds in obtaining consent to a meal. When he fails, artificial feeding must be employed; if the patient loosens his jaws, the catheter can be introduced by the mouth, but if he does not, then the nasal tube must be passed.

Hydrotherapy must be used with caution. Cold douches are of no value for patients who have no power of reaction, they can only be used for simple depression, or in convalescence. Hot douches, and especially hot baths, are preferable; they dilate the peripheral vascular system and increase the flow of urine. Short baths (a quarter-of-an-hour or half-an-hour) are best for depressed patients. For those, who react to anxiety by motor agitation, prolonged baths of two to three hours should be ordered.

The medicinal treatment consists of tonics, such as quinine and extract of kola, and stimulating injections which raise arterial tension (artificial serum). If the patient has arterio-sclerosis, milk diet (absolute or mitigated), iodide of potash, and tri-nitrine for the paroxysms of anxiety, can be used; constipation must be overcome by laxatives, saline purgatives, podophyllin, or laxative powders.

Insomnia can be relieved by hypnotics, and the best among these is a hot bath, kept up for two or three hours at the end of the afternoon; a quiet sleep of some duration results. Chloral, sulphonal, trional, etc., only take a second place. Anxiety is greatly relieved by the use of opium in conjunction with rest in bed and prolonged baths. Masselon prescribes:—

℞. Ext. Opii. - - - - - - gr. xv.
Glycerini - - - - - - ʒj.
Aq. Destill. - - - - - ad ℥iss.
Misce. Ft. Mist.

The first dose of this is ♏xij. (containing gr. ¼ of extract of opium), the next dose is double this quantity, then thrice, and so on until the therapeutic effect is obtained, giving, if necessary, as much as ℥ss. If the condition of anxiety gets less, progressively decreasing doses are then given. The remedy is given by the mouth, but, if hypodermic injections are used, morphia is preferable. Opium cannot be looked upon as the specific remedy for melancholia, but it brings the patient into a condition such that the efficacy of the other part of the treatment appears to be more marked.

In the chronic forms, a condition of intellectual impairment, more or less profound, eventually supervenes, and the decay of the patient prevents his return to active life. Placing him in domestic colonies, asylum-colonies, such as exist in Germany, is one of the best measures which can be adopted as it removes the unfortunate subject from contact with clamorous and quarrelsome madmen.

THE USE AND ABUSE OF DIGITALIS.

Pfaff, in the *Boston Medical and Surgical Journal*, states that the therapeutic effects, which we seek to obtain from digitalis, are increase of blood-pressure, increase of the volume and decrease in the rate of the pulse. As soon as the rate begins to increase, it is an indication that the danger of over-stimulation has been reached. It must, however, be always borne in mind that, in some patients, the vagus does not respond to stimulation by digitalis, consequently no reduction in the pulse-rate occurs. In such cases, we cannot rely on counting the pulse only to show if the drug is acting. In other instances decrease in rate may counterbalance increase in pressure, so that, in a given time, the heart may be doing a less amount of total work than in ordinary cases. In other instances again, the heart's action may be already toxically influenced, and the beats so much increased in number, that the pulse waves have no time to reach the radial artery. Auscultation is, therefore, quite as important as counting the pulse, for, if neglected, in such cases as those just mentioned, the poisonous paralysing effect of digitalis on the circulation may take effect before a rise in the pulse-rate occurs to give warning. The main action of digitalis is to raise the blood-pressure, and thus ensure better-filled arteries. It may therefore, be used in all cases in which the pressure is too low, and in all conditions, in which the symptoms point out an incomplete filling or emptying of the circulatory system. First of such conditions to be mentioned are all valvular lesions with failing compensation. It is a gross abuse to give the drug when compensation is well established, but this is often done.

The best results are obtained in mitral and in aortic insufficiency; in the latter, a larger dose is necessary than in the former to get a good effect. The effect is not so marked, as a rule, in mitral stenosis and in recent valvular lesions. It may be given, but always very cautiously and with constant watchfulness, in myocarditis, dilatation of the heart, palpitation, and pericarditis. The palpitation must not be caused by dyspepsia or general nervous debility. Experiment has recently proved that, if blood containing digitalis is, by means of the coronary artery, sent through an irregularly-beating isolated heart, the beats become regular again.

Its use as a diuretic is only indicated when the lack of diuresis is clearly due to disturbance of the circulation, and this same factor is present in all other diseases, in which its use has been found to be of benefit, such as pneumonia, emphysema, etc. (*Therapeutic Gazette*, Sept. 1906.)

TREATMENT OF WARTS AND PAPILLOMATA OF THE SKIN.

Lecturing recently at the Hôpital Saint-Louis, Brocq recommended electro-cautery or scraping, as the quickest and most certain method of treatment of papillomata of the skin and some warts. The method is rather painful, and, if it is declined, the following may be substituted:—Every second day, the coating of salicylic collodion, which has been applied two days before, is removed with ether. Then all the horny epidermis which has been blanched by the collodion, is rubbed off by means of a flame-sterilised scraper, or a piece of pumice-stone. After cleaning, the base of the papilloma is touched carefully with nitric acid, or preferably with glacial carbolic acid. A coating of 10 per cent. salicylic collodion is then applied; salicylic plaster of the same strength may be used, but the collodion sticks on better, especially on the hands. This procedure is repeated every two days so long as necessary. If the skin gets inflamed, the part must be covered with zinc ointment, and the treatment resumed when all symptoms of inflammation have been subdued. (*Journal de médecine et de chirurgie.*)

AN EASY METHOD FOR REDUCING A PARAPHIMOSIS.

Nemery describes, in the November number of the *Archives médicales belges*, an ingenious method for obtaining an easy reduction of paraphimosis. The end of the penis, including the strangulating band, is wrapped round with a layer of absorbent wool, soaked in a mixture containing equal parts of a solution of adrenalin ($1^\circ/_{oo}$) and a 10 per cent. solution of cocaine. This is kept in place for about a quarter-of-an hour by a few turns of bandage, applied with a moderate amount of compression, so that the glans and the œdematous part of the prepuce may be thoroughly acted upon by the solution. On removal, the œdema will be found to have almost completely disappeared, and very little traction is required to bring the prepuce forwards into its place.

FOR THE INSOMNIA OF OLD AGE.

The following is included, by the *Gazette médicale de Paris*, in its *carnet du praticien* :—

℞.	Ext. Cannabis Ind. Liq.	♏ vj.
	Codeinæ	gr [illegible]
	Syrup. simpl.	ad ℥ss.

Misce. Fiat haust. h. s. s.

The effect of the cannabis is increased by giving the patient a hypotensive drug, such as tincture of aconite, or tincture of veratrum viride, in three-minim doses three times a day.

OILY COLLYRIA.

It is claimed, by Panas and Scrini, that collyria made with oil possess great advantages over the usual watery preparations. Application is easy, and tolerance is remarkable; action is quicker, more intense and more prolonged, because the oil remains a longer time in the conjunctival cul-de-sac; instillation produces neither spasm of the eyelids, nor lachrymation, nor does an oil collyrium of cocaine cause exfoliation of the corneal epithelium; they are stable and do not alter in composition (eserine, for instance, does not change into rubr. eserine, which is so particularly irritating to the conjunctiva); they are aseptic, and remain so for an indefinite period.

For the preparation of these collyria, the olive oil used must be specially cleansed and sterilised. First of all it is washed with strong alcohol to remove any fatty acids. For this purpose, it is mixed with half its volume of 95° alcohol, and the two are left in contact for some days, being shaken up from time to time. The mixture is then transferred to a vessel fitted with a tap below, by means of which the oil is drawn off, when the two fluids have separated into an upper and a lower layer. It is, as a rule, perfectly limpid, but if any impurities are seen in suspension, or if the oil appears to be not quite clear, it must be passed through a filter-paper. For sterilisation, it is sufficient to place the vessel holding the oil on a sand-bath, heat the oil to 120° C., and keep it at this temperature for about a quarter-of-an hour. With a sterilised plug in the mouth of the bottle, the oil will keep fit for use indefinitely.

Scrini recommends the following formulæ:

1.	℞.	Cocain.	gr. iij.
		Ol. Olivæ (Steril.)	ʒiij.
2.	℞.	Eucain. β	gr. v.
		Ol. Olivæ (Steril.)	ʒiij.
3.	℞.	Holocain.	gr. iss.
		Ol. Olivæ (Steril.)	ʒiij.
4.	℞.	Stovain.	gr. iij.
		Ol. Olivæ (Steril.)	ʒiij.

MYDRIATICS.

1. ℞. Atropin. - - - - - gr. [illegible] v. [illegible].
Ol. Olivæ (Steril.) - - - - ʒiij.
2. ℞. Duboisin. - - - - - gr. [illegible] v. [illegible].
Ol. Olivæ (Steril.) - - - - ʒiij.
3. ℞. Homatropin. - - - - - gr. [illegible] v. [illegible].
Ol. Olivæ (Steril.) - - - - ʒiij.
4. ℞. Scopolamin. Bromohydrat. - - gr. [illegible] v. [illegible].
Ol. Olivæ (Steril.) - - - - ʒiij.

To prepare the last, the scopolamine must be dissolved in a little alcohol before being added to the oil. After well mixing, the oil must be warmed to 40° C. in order to drive off the alcohol by evaporation.

MYOTICS.

1. ℞. Eserin. - - - - - gr. iss. v. [illegible].
Ol. Olivæ (Steril.) - - - - ʒiij.

A high temperature changes eserine into rubr. eserine, and Scrini recommends that the eserine should be dissolved in æther, so as to form a 1 per cent. solution before mixing with the oil. The æther is then driven off by standing the mixture in a bain-marie at a temperature of 45° C. The resulting 1 per cent. collyrium is clear, transparent, and will not show any alteration of colour for many years.

2. ℞. Pilocarpin. - - - - - gr. iij.
Ol. Olivæ (Steril.) - - - ʒiij.

(*Journal de médecine et de chirurgie prat.*)

FIBROLYSIN.

Two articles, in the December *Therapeutische Monatshefte*, testify to the good results obtained from the use of this mixture of thiosinamine and salicylic acid. Salfeld, of Wiesbaden, reports on some cases of arthritis deformans with contractures, all of which were very considerably benefited, and Schourp, of Danzig, gives details of some old-standing strictures of the urethra. Merck's ampullæ were used, each of which contains 2·3 c.c. of a sterile solution of fibrolysin, corresponding to 0·2 g. (gr. iij.) of thiosinamine. This fluid was injected subcutaneously in the gluteal region, and gave rise to a slight sensation of burning which passed off very quickly. No general after-effects were observed, and little or nothing at the site of the injections, which were given about once a week.

The results obtained, with very few exceptions, in a large number of cases of arthritis deformans and chronic polyarthritis treated by Salfeld, were:—Speedy reduction of exudation or swelling, lessening of the crepitations, disappearance of the pains, and, above all, much better power of movement in the affected joints. It must be noted, however, that all these patients were undergoing the Wiesbaden "cure" at the same time, but Salfeld does not hesitate to attribute his, in some cases, surprising results to the action of the fibrolysin.

Notes by the Way.

Economic Aspects of Medical Practice.

THE eternal question of hospital abuse is at present going through one of the periodical phases of acute exacerbation which diversify the course of a chronic grievance of the medical profession. It can scarcely be said, that the recent discussions have thrown any new light on the subject or helped to bring us within sight of a satisfactory solution of the problem. The passage of resolutions declaring that excessive medical charity is an evil, and that hospitals are misused, may relieve the feelings of a meeting, but has no appreciable influence on public opinion. Nor is the reason far to seek. To the people at large the matter is only a doctors' grievance, and for this view, wrong as it is, the profession has mainly itself to thank. Beneath the surface of altruism there runs an undercurrent of assumption—tacit and perhaps unconscious, but none the less active—that the public is under a special obligation to the profession which gives every practitioner a right to a living wage. It would be well if it were clearly recognised that doctors have no better claim to the patronage of the public than bakers or butchers. This may seem a hard saying, but it is better to face a bitter truth than to fool oneself with a delusion. Medical practice is subject to the same rigid economic laws as every other calling in which man earns his bread by the sweat of his brow. It is a struggle in which the race is to the swift and the battle to the strong, and in which the weak are thrust aside and fall by the way. Collectivism may mitigate the fierceness of the struggle, but it cannot alter conditions that spring from the fabric of human nature. Medicine has the immense advantage of many other avocations that it is not a luxury but a necessity. This does not mean, however, that every man who chooses it as a profession is necessary. He has to justify his existence by his work, and if he cannot do this for himself, neither the General Medical Council, nor the British Medical Association, nor any union or committee, can do it for him.

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The Abuse of Hospitals.

IT is, of course, undeniable that hospitals are to some extent abused, or, as we are now invited to say, "misused"—one of those distinctions without a difference dear to the mind of the pedant. But on the whole there is not much to complain of on this score. Many hospitals have now almoners and inquiry officers, who weed out undeserving cases pretty thoroughly. The recent discussions revealed a wide divergence of view between hospital staffs and general practitioners in regard to the out-patient department. The plain truth is their interests in this matter are irreconcilable. The doctor who practises within the sphere of influence of a great hospital naturally looks upon the people who crowd the out-patient department as bread taken out of his mouth. On the other hand, the hospital physician or surgeon looks upon them as material available for teaching purposes. The patients care for neither of the conflicting interests, but go where they think, rightly or wrongly, they will be most efficiently and quickly healed. If they get the treatment they need for nothing, can they fairly be blamed for taking advantage of the offer? The outcry against the out-patient departments is open to the suspicion of being just a little artificial. A considerable proportion of the patients who go to the hospital have been under the hands of one or more outside practitioners; not a few indeed are sent there by doctors unwilling to take the responsibility of an obscure or difficult case. Sometimes, if the truth must be told, the out-patient room is the receptacle of the "squeezed oranges" of general practice. The proposal that no one should be seen as an out-patient unless on the recommendation of a general practitioner, would do little to mitigate the evil, for it is the general practitioners themselves who are to a large extent accountable for its existence. Unscrupulous patients could easily find a doctor, who, for a fee, would give them the required recommendation. On the other hand, the hospitals are compelled by the very law of their being to throw their doors as wide open as they decently can. It is only in the out-patient department that the student has an opportunity of learning "the daily task, the trivial round" of the duties that will fall to him as a general practitioner. There is much complaint at the present day that men leave the hospitals who know all about rhinoscleroma, but find themselves at a loss when confronted with a case of "red-gum." The abolition of the out-patient department, which is advocated by some perfervid spirits, would increase this evil a hundred fold. Real abuse of hospitals should of course be strictly guarded against. It was suggested by THE PRACTITIONER years ago that warning notices should be put up in the waiting rooms of all hospitals, intimating that all persons, well able to pay for medical treatment, who took advantage of charity intended solely for the poor, would be prosecuted for fraud. This, we are convinced, would effectually prevent any misuse of hospitals.

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Doubtful Remedies.

THE remedies for the hospital grievance that have recently been proposed are all more or less impracticable. Compulsory insurance would secure the practitioner's fees, but it could not force the person insured to seek counsel of him. And how is insurance to be compelled? The free and independent Briton will not, like the submissive Teuton, go in State-leading strings. Our sons of labour are so much accustomed to have their needs provided for out of the public purse that they are not at all likely to be willing to pay for what they can get for nothing. A public medical service would mean contract practice writ large, with all its inherent evils immensely aggravated. The municipalisation of hospitals would deprive patients of the vast benefits to them that arise from a system, which makes every physician and surgeon in his own interest do his utmost for those under his care, and constantly strive to perfect his skill, and discover better methods of treatment. If hospital staffs are put under the heel of Bumble, these appointments will cease to be sought by the men most fitted for them, and will become the prizes of intrigue, not of merit. Municipal control would degrade the profession and hinder the progress of science. State control might be less objectionable, but the examples of hospitals in foreign countries tend to show that patients are not so well off under a government administration as they were under our own voluntary system.

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Medical Peris and the Surgical Paradise.

REFERENCE was made in the February number to the fact that women are again striving to effect an entrance into the Royal College of Surgeons of England, which still keeps its portals obstinately closed against them. It appears that there is a majority among the members of the Council in their favour. The Fellows, too, are believed to be generally not hostile. But it is hinted that the rulers of the College have the fear of the Members before their eyes, and intend to have recourse to a referendum on the matter. If this is the case, the situation is decidedly curious. The past history of the College has not accustomed us to the exhibition, on the part of the Brahminical caste which is supreme in its councils, of any very tender solicitude for the feelings and opinions of the mere Member. But in this instance the Member is thought, with what justice we do not know, to be prepared to declare *Nolumus leges Collegii mutari.* To the profane mind, therefore, the referendum looks like a somewhat disingenuous attempt to shift the responsibility of a decision to the shoulders of those who have hitherto been denied any share in the management of the College. It appears likely that the Peris of the London School of Medicine for Women will still have to wait disconsolate at the gate of the Paradise in Lincoln's Inn Fields.

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Women and the Medical Profession.

In our last issue we quoted the unfavourable opinion recently expressed by His Excellency Professor Ernst von Bergmann as to the fitness of women for the profession of medicine. It is only fair, therefore, that we should give the entirely different view held by one whose opinion will, in this country at least, probably carry greater weight than that of the distinguished German surgeon. Speaking at a meeting of the New York Women's Association held in memory of the late Dr. Mary Putnam Jacobi, Professor Osler is reported to have said that he had long been awaiting the advent of a woman physician so commanding that she will rank with Harvey, Pasteur, and Virchow, and he added that he had not the slightest doubt that such a one would arise. The learned Professor went on to say that, while a larger proportion of women than men were unfit for the practice of medicine, there was a relatively large percentage of women fitted for scientific work. It was encouraging, he added, to see so many women pursuing laboratory researches here. They might meet men as equals, since what they lacked in initiative and independence was counterbalanced by a more delicate technique, a greater patience with minutiæ, and a greater mastery of detail.

Even when the necessary discount is made for the fact that Professor Osler was addressing an association of medical women, this is a remarkable utterance. We cannot say that we agree with it, but the ladies will naturally prefer Professor Osler's opinion to ours.

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The Noise Nuisance.

THE deputation from the Betterment of London Association which asked the Home Secretary to do something to abate the concourse of anything but sweet sounds, which make our streets places of torment to any but the deaf, got little encouragement from Mr. Gladstone. The motor bus has come to stay, and it can only be hoped that, as the machinery is perfected, it will cease to cleave the general ear with horrid din. London used to strike the foreign visitor by its silence; this particular charm it must be content to lose, at least for a time. Meanwhile, New York and Boston have set a good example by stopping the whistling and shrieking of the tugboats that have hitherto made night hideous. An American writer is so penetrated with the evil of noise that he has called upon all churches throughout the States to bind over their communicants to the following behests of applied Christianity:—

1. Not to cart sheet iron through the streets at night. 2. To stop the barking of dogs. 3. Not to blow factory whistles at six in the morning. 4. Not to practise on wind instruments. 5. Not to keep the gramophone going for more than eighteen hours at a stretch. 6. Not to murder sleep with locomotive whistles. 7. Not to drive noisy milk carts. "If Norway," it is asked, "can have quiet ones, why not America?" 8. Not to pave streets with din-producing granite blocks.

From some of these sources of noise London is free. Nevertheless we suffer from a vast amount of needless noise, which is a serious obstacle to the recovery of the sick, and to many of those who are well a disturbing influence, which may interfere with the delicate adjustments and co-ordination of nervous centres which make efficiency in work.

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The Medical Profession in Germany.

It is not only good "to see ourselves as others see us," but to see others as they see themselves. It is pretty widely believed that the lot of the German doctor is a happier one than that of his British brother; and, in particular, that the Fatherland is, in matters medical, under the beneficent sway of science, and that the demon of quackery is chained up so fast by the law as to be harmless. A real state of things is revealed by Professor Kosmann of Berlin. The German State Examination gives the title of "approved practitioner," but scarcely anything else. The practice of medicine is as free in Germany as in our own enlightened country, and quacks may call themselves "practitioners," provided they take care to add the words "not approved." Foreigners can practise without going through any legal or academic ceremony, and can even use the title "Doctor" if they indicate the source of their degree. The number of quacks is enormous, and qualified practitioners suffer severely from their competition. Moreover, the stress of competition among the doctors themselves is very great. Contract practice especially in connexion with the system of compulsory insurance against sickness, weighs heavily on the profession; although the fees are very small, there is an eager scramble for them. Against the sea of troubles that threatens to overwhelm them, the German doctors have taken arms by forming associations for the protection of their professional interests. These local forces are co-ordinated in a central union, the *Deutsche Aerztevereinsbund*. There are also throughout the Empire medical councils, "chambers," elective bodies in which there is a representative for every fifty practitioners. These "chambers," as they are called, are the official voice of the profession in each province; they are, moreover, the guardians of the dignity of the profession, having the power to form courts of honour, which can adjudicate on cases of what is called here "infamous conduct in a professional respect." Effect is given to the decision of these courts by the civil power. The profession can therefore deal with its own backsliding members. But against quackery, it is almost helpless. On the whole, it would seem that, in the matter of professional rights and privileges, we are not much worse off than our German brethren.

Reviews of Books.

Nervous Diseases in Childhood and Early Life. By JAMES TAYLOR, M.A., M.D., F.R.C.P., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, Queen Square; Physician to the North-Eastern Hospital for Children, and to the Moorfields Eye Hospital. London: J. & A. Churchill.

DR. TAYLOR'S book is an extremely well-written and mostly up-to-date account of nervous diseases as met with in children and in the earlier years of puberty. A book of this nature, which does not aim to be a text-book of diseases of the nervous system, must somewhat overlap the territory usually allotted to the ailments of adult life, unless some forms of disease, which but rarely affect the child, are to be omitted altogether. Yet we think it scarcely advisable to devote eighteen pages to the discussion of Syringomyelia, a disease which practically never shows symptoms before the age of fifteen, and usually much later. Many of these pages deal with the morbid anatomy and pathogeny of this curious ailment, and illustrations of sections of the diseased cords are given. We recognise here a tendency of the author to unduly enlarge upon a pet theme, and to quote overfully from previous work of his own upon this subject. Yet, in spite of this lengthy description of the morbid processes, not a word is said as to the class of patient attacked by the disease, nor at what age the symptoms appear. The only clues given are two, a picture of a man, who appears to be about 35, with his tongue out, and a reference to another patient, who was in the habit of severely, though unconsciously, burning his fingers when smoking cigarettes. These sole cryptic allusions to the age of the patients, who may suffer from Syringomyelia, do not suggest either childhood or early life. Moreover, no mention of its occasional association with cheiromegaly, or acromegaly, is made, nor is perforating ulcer included under the list of trophic symptoms which it may produce.

Dr. Taylor gives a full and interesting chapter on Infantile Hemiplegia, and classifies it in three natural groups: (1) the pre-natal cases due to endarteritis and thrombosis from maternal syphilis; (2) the cases occurring at birth, and due to injury; and (3) the post-natal cases due to encephalitis. It is this latter group which Dr. Taylor states to be the sole important cause of hemiplegia of sudden onset in young children, and he dismisses, with, we think, too scanty reference, such causes as cerebral hæmorrhage in whooping cough, and thrombosis after typhoid, or diphtheria. Acute encephalitis is undoubtedly a much more frequent disease than was formerly recognised, and, being an acute inflammatory lesion followed by degeneration of the cortex, it is comparable up to a point with acute anterior poliomyelitis. Encephalitis is, however, by no means limited to the motor cortex, and it may, and does, attack the frontal, occipital, or cerebellar cortex, producing the symptoms referable to disease of these areas rather than hemiplegia. An error, which we should not have expected to find here (p. 63), gives von Bechterew instead of Nothnagel as the original describer of paralysis of the emotional, with retention of the volitional, movements of the face in lesions of the thalamic region. A good description is given of the clinical aspect of the Myopathies, though the three pages, devoted to the history of the disease, are practically copied, the wording being here and there slightly altered, from Gowers' and Taylor's *Manual of Diseases of the Nervous System*. An excellent account also is given of that rare disease Family Periodic Paralysis, yet no mention is made of the spinal type of progressive muscular atrophy found in young infants, which is associated with the name of Werdnig-Hoffmann.

The chapter on Meningitis is good, but Dr. Taylor omits almost all mention of the work that has of late been done in identifying the post-basic form with epidemic cerebro-spinal meningitis. Nor, in distinguishing clinically the post-basic form from tubercular meningitis, with which it is most likely to be confounded, does he refer to lumbar puncture. He gives his decision that, as a curative procedure, it has little or no value, with which most physicians will be inclined to agree. Lumbar puncture may, however, be most useful in distinguishing the two forms of meningitis, the fluid in the tubercular form being almost invariably clear and sterile, with a cell deposit mostly of lymphocytes, while, in the post-basic variety, the fluid withdrawn is usually turbid, the cellular deposit containing an excess of polymorphonuclears, and often the diplococci of Weichselbaum can be grown by culture.

A very rare disease, recurrent third nerve paralysis and its associated headache, is described at length, but true migraine, which so frequently begins in childhood, and is so liable to be mistaken for biliousness and all sorts of gastro-intestinal disorders, is left undescribed.

In the article on Diphtheritic Paralysis, alcohol is mentioned as "extremely useful" in the treatment, a remedy which some, at all events, would hesitate to use, and even consider to be contra-indicated. Indeed, it does not seem probable that, in a condition where there is already peripheral neuritis, with fatty degeneration of the cardiac muscle of toxic origin, alcohol could be anything else than harmful. The use of bromides, too, in the treatment of the vomiting of diphtheritic paralysis, is not referred to, though no other treatment for this complication is suggested.

Rather strange is the entire omission of a description of myelitis, the word not even appearing in the index. Yet this disease is not so very rare in children, especially as a sequela to some of the infections, as typhoid and measles. A short chapter is, however, devoted to the description of Achondroplasia, which, in no sense of the word, is a disease of the nervous system. Amaurotic family idiocy is well described, as are microcephaly, mongolism, and cretinism; but oxycephaly, with its associated optic atrophy, is not referred to.

The style of the book is most readable, and the clinical knowledge displayed is mostly of a high order and extensive. It is, therefore, in no carping spirit that we have drawn attention to several points that, we think, call for re-arrangement, but rather in the full belief that this volume will prove a success, and that Dr. Taylor will be called on to produce a second and improved edition.

Reference Handbook of the Diseases of Children. By Professor F. FRÜHWALD. Edited with additions by T. S. WESTCOTT, M.D., Associate in Diseases of Children in the University of Pennsylvania. With 176 illustrations. London: W. B. Saunders Company. 18s. net.

This ponderous volume is the result of twenty years' study, so the author states in his preface, and is on that account entitled to careful consideration. It is arranged on the dictionary system, a method which involves the disadvantage that some pages consist very largely of cross-references. This American edition is well printed and well illustrated. But having said thus much, we find it difficult to say much more in its favour. There is the same lack of proportion which is noticeable in so many text-books purporting to deal specially with diseases of children, for instance, more space is devoted to exophthalmic goitre, a condition almost unknown in childhood than to spasmus nutans, a condition peculiar to children. Even on some of the most characteristic diseases of childhood the information is not merely scanty but inaccurate; for instance, for infantile scurvy the only treatment for infants is "pastearised or even unboiled milk," while fruit juice and vegetables are mentioned as suitable only for older children. Rheumatic nodules are described as a "form of joint disease, which frequently follows acute rheumatism"; they are said to be painful to pressure, and liable to leave permanent contractures of the tendons. Even the illustrations, of which many are excellent, are not altogether trustworthy. Under the head of cretinism, at least one of the illustrations represents a typical Mongol imbecile, and as we can find no reference to Mongolism anywhere, we conclude that Dr. Fruhwald has not yet distinguished between a Mongol and a cretin, a very important distinction from the prognostic and therapeutic standpoint. Anyone who attempted lumbar puncture by following the illustrations given in this work, would fail to enter the theca altogether, as the trochar in both figures is represented as sloping away from the middle line.

Apart from errors of observation, there are statements in which even his editor has been unable to reproduce Dr. Fruhwald's views without some qualification, as they are contrary to common experience. For instance, the author says that in "every condition designated as a teething disease, it is possible to find some other ætiologic factor." "Lancing of the gums," he says, "has no value whatever." The editor adds that "most American physicians will refuse to accept this dictum." Again, the author devotes a whole section to "growing-pains," and their treatment by intestinal antiseptics, and it is left to the editor to point out, what one would have thought was familiar to every medical man, with even a small experience of children, that "growing pains" usually mean acute rheumatism. It is difficult to understand what reason there could be for translating such a work when American pediatrists have themselves produced so many far superior text-books.

Practical Notes.

Improper Feeding of Children.—In a valuable paper published in the *International Clinics*, by Mr. Edred M. Corner, he gives an account of the more important results of the improper feeding of children, which come more particularly under the notice of the surgeon. Two of the diseases, more commonly met with in young children as the result of improper feeding, are *scurvy* and *rickets;* but it must be remembered that diarrhœa is one of the most frequent symptoms of local intestinal trouble, due to the presence of irritating material in the alimentary canal. The results of improper feeding may be due either to the fact that the child is suffering from some derangement in health, which prevents it from digesting its food, or to the improper food which is administered to the child. Occasionally, however, the fault lies both with the digestive power of the child and with the food. Mr. Corner points out that there are three lines to be followed, in considering the consequences of food, proper or improper, which ferments within the bowel, namely, (1) the effect of the toxins which are produced there; (2) the effects of the gases generated; (3) the effect of the fermentation in the alimentary canal on the bowel itself. The toxins, which are produced in the bowel, are absorbed by the wall of the intestine, and are carried by the portal vein to the liver; hence follows liver enlargement; and by the lacteals from the intestine to the receptaculum chyli, thence into the thoracic duct, and thus the poison gets into the general blood stream in two ways. "The child is querulous, often crying and not taking its food well, it does not sleep well, its pillow is often wet from perspiration about the head, its bowels are inactive or loose, its abdomen is large and may show multiple hernias, divaricated recti, and bulging flanks; it suffers from wind, and the stools are offensive." At this stage of the trouble, the diet must be regulated both as regards quality and quantity, and the bowels regularly evacuated by the following mixture:—

R.	Sodii Bicarbonatis - - -	gr. vj.
	Infusi Rhei - - - -	ʒj.
	Elixir Simplicis - - -	♏xvj.
	Aq. - - - - ad	ʒiij.

The medicine to be taken three times daily before food;

or the following:—

R.	Pulv. Rhei - - - -	gr. i.
	Sodii Bicarbonatis - - -	gr. i.
	Syrupi Zingiberis - - -	♏vij.
	Aq. Menthæ Pip. - - ad	ʒj.
	Ft. Mist.	

One teaspoonful to be taken three times a day before meals.

If the toxins are not effectually eliminated from the body, they attack the growing ends of the long bones and the periosteum, causing rickets; in addition, the toxins attack the blood-producing organs of the body such as the red marrow of the bones, the spleen, and the vascular system generally, producing the condition known as scurvy rickets. The effects of the gas formation, produced by fermentation, are to distend the intestines, and consequently to increase the intra-abdominal pressure, which must of necessity affect the weaker portions of the anterior abdominal wall; these particularly are the linea alba, umbilicus, and the inguinal regions, and, if the gas formation is large in any of these regions, hernial protrusions may appear. Moreover, though herniæ may not appear at this time, the prolonged distension will cause the external and internal oblique muscles of the anterior abdominal wall to become permanently stretched and weakened, so that bulging in the flanks occurs. If this condition persists in the adult, it no doubt predisposes to the appearance of herniæ.

The decomposition and fermentation of the food in the alimentary canal lead to gastritis, enteritis, colitis, and sometimes to jaundice. These catarrhal conditions of the bowel predispose to infection with tubercle bacilli, and tabes mesenterica follows. The irritable condition of the gut may lead to intussusception and prolapsus recti. If the inflammation spreads to the wall of the intestine, the gut loses its tone and constipation follows. If it spreads to the peritoneum, which covers the bowel, fluid accumulates in the peritoneal cavity, and, should there be a patent processus vaginalis present, a hydrocele of the tunica vaginalis forms.

At the end of his paper, Mr. Corner sums up as follows:—
1. The best foods, unwisely administered, can produce symptoms of improper feeding. These symptoms may often be seen in breast-fed babies. 2. The fault may be with the child, and not

with the food. 3. At birth, the alimentary canal is sterile. With the admission of organisms, begin the decomposition of food residues and the intestinal education of the child. 4. There is a recognisable stage of toxæmia before any rickety signs are manifest. Treatment should be adopted then. 5. The toxins are known to produce gross changes in some organs; probably they do so also in other structures as yet unknown to us. 6. The gas formation may produce inguinal and umbilical herniæ, divarication of the recti, a large abdomen, bulging flanks, hydroceles, etc.; sowing seeds of weakness for the possible future development of herniæ, varicoceles, hydroceles, spermatoceles, etc. 7. In the intestine itself many changes may occur such as: gastritis, enteritis, appendicitis, colitis, intussusception, prolapsus recti, etc. 8. Adenoids are commonly a sign of rickets or improper feeding. 9. Congenital syphilis is held responsible for many rickety changes.

ADENOIDS.—Upon the roof of the naso-pharynx, extending down the posterior wall of the pharynx as far as the level of the anterior arch of the atlas vertebra, and extending laterally as far out as the orifices of the Eustachian tubes, is a collection of adenoid or lymphoid tissue called the *pharyngeal tonsil*, or Luschka's tonsil. Hypertrophy of this lymphoid tissue constitutes the condition known as *adenoids*, the harmful effects of which are chiefly due to their interference with nasal respiration. There is an orifice upon the centre of the pharyngeal tonsil, which leads into a small recess, into which the orifices of many mucous glands open. In the paper, already quoted above, Mr. Corner points out that just as rickets affects the bones of the vault of the skull, so it most probably produces bony deformation in the base of the skull, the bones becoming thickened and later sclerosed. The naso-pharynx will now become affected, and the child will consequently experience discomfort in breathing through the nose. Adenoid growths in the naso-pharynx seriously interfere with the development of the nasal cavities, the palate, and the alveolar arch; as a result, the arch of the palate becomes narrow and high, and the upper jaw becomes somewhat compressed. If a negative pressure is produced during inspiration, the nasal and palatine bones will fall in, causing the child to breathe through the mouth instead of through the nose. In the unventilated pharynx, micro-organisms readily flourish ; this causes local irritation of the lymphoid tissue, which naturally occurs there, giving rise to a condition recognised as adenoids. The naso-pharynx, stuffed with hypertrophied lymphoid tissue, ceases to develop, mainly in consequence of disuse. It will thus be seen that improper feeding leads to rickets, and rickets may lead to adenoids. "If one keeps in mind the stage of intestinal toxæmia before rickety changes have already declared themselves in the bones, one will realise how often cases, sent to hospitals as cases of adenoids, merely want proper dieting and general treatment, to be followed by a course of nasal respiratory exercises, and do not require operation at all. Knowing that obviously rickety bones become straight as the result of treatment and healthy growth, the proper treatment must be adopted, and time allowed to elapse for the rickety changes in the bones of the naso-pharynx to be removed, before a final prognosis can be given. Certain it is that many of these children, treated in this way, avoid the need of all operative treatment."

It must be kept in mind, however, that, if the adenoids are left untreated, and especially if the pharyngeal tonsils are enlarged too, the patient is very liable to repeated attacks of bronchitis and even bronchial asthma. Middle-ear catarrh and deafness frequently accompany and complicate chronic enlargement of the tonsils and neighbouring adenoid tissue. Tuberculosis of the cervical lymphatic glands is, no doubt, a result of chronic enlargement of the pharyngeal and faucial tonsils.

TREATMENT OF ADENOIDS.—In the slighter cases of adenoids, with some enlargement of the tonsils, a weak alkaline lotion, such as the following, should be syringed through the nose and fauces so as to free the lymphoid tissue, so far as possible, from micro-organisms, and to prevent crusts forming upon the surface :—

℞.	Sodii Bicarbonatis	gr. v.
	Boracis	gr. v.
	Sodii Chloridi	gr. ij.
	Glycerini	ʒi.
	Aq.	ad ℥i.

Ft. Lotio.

Astringents, such as the following, may be painted on the tonsils and the adenoid tissue:—

R.	Aluminis	ʒij.
	Tannin.	ʒss.
	Glycerini	℥ss.
	Aq. Rosæ	ad ℥ij.
Ft. Pig.		

Or,

R.	Iodi	gr. ij.
	Potassii Iodidi	ʒss.
	Glycerini	ad ℥j.
Ft. Pig.		

Tonics should be administered, such as the phosphate of iron or steel wine. If the patient is no better for the treatment, and the symptoms of enlarged tonsils and adenoids still persist, then these growths should be removed. A well-known authority says:—"I believe they (adenoid vegetations) are present in *many*, and cause symptoms only in the *few*: and that the question of their removal should be determined, not by their presence alone, but by the evidence of obstruction of the nostrils, deafness, recurrent 'colds' in the throat, distortion of the chest, and enlargement of the cervical glands."

THE LYMPHATICS OF THE CONJUNCTIVA.—The lymphatics of the conjunctival sac form, like the conjunctival veins, pretarsal and retrotarsal networks, which communicate with each other through the tarsal plates. The lymph from the outer part of the conjunctival sac drains chiefly into the pre-auricular and parotid lymphatic glands, hence any infective process, which occurs in this part of the conjunctival sac, such as septic phlyctenulæ, styes, or a primary chancre, will cause an enlargement of the glands already mentioned. Such swelling is sometimes mistaken for a displaced interarticular fibro-cartilage of the temporo-maxillary articulation. The lymphatics, which drain the inner portion of the conjunctival sac, travel along with the facial vein, and open into the submaxillary lymphatic glands, which may be readily felt just under the lower border of the mandible. The enlarged lymphatic glands in this neighbourhood must, of course, be differentiated from chronic enlargement of the submaxillary salivary gland itself.

STYES.—A stye, or hordeolum, is a circumscribed acute inflammation situated at the follicle of an eyelash, or in the cellular tissue of the eyelid itself. It commences as a hard swelling, with some tumefaction and local œdema of the connective tissue of the eyelid, often with some chemosis of the ocular conjunctivæ, the latter is more especially noticed if the stye is situated near the outer canthus. The preauricular lymphatic glands may frequently be felt to be enlarged, and are oftentimes tender. As a rule, the acute circumscribed inflammation ends in suppuration; then a small yellow area may be seen at the most prominent part of the swelling, which is usually at the margin of the eyelid. Styes occur at all ages, but they are more common in children and young adults, and they often appear in crops. As a rule, the patient is out of health, and suffering from constipation, acne spots, or errors of refraction, such as hypermetropia, or hypermetropic astigmatism. Until suppuration actually occurs, hot boric acid fomentations should be used, and the patient purged. When suppuration has occurred, the eyelash, which is usually in the centre of the yellow area where the pus is pointing, should be pulled out, and then, if necessary, the swelling should be incised, and again hot boric acid fomentations applied. Syrupus Ferri Phosphatis, in drachm doses, should be given twice or three times daily after food. Calcium sulphide, in doses varying from $\frac{1}{8}$ to $\frac{1}{2}$ a grain for an adult, given twice daily, has been recommended in cases of recurrent styes. When the more acute inflammatory symptoms have disappeared, the following ointment may be prescribed:—

℞. Unguenti Hydrargyri Oxidi Flavi - pt. j.
Vaselini - - - - pt. ij.
Ft. Ung.

A small piece of the ointment to be applied to the margins of the eyelids with a fine camel-hair brush night and morning. All errors of refraction must be corrected by the use of appropriate glasses. A generous diet, plenty of open air exercise, and, if possible, a change of air are also indicated.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.—In reply to many correspondents, we have to state that this subject, including the treatment, was fully dealt with in our August 1906 number, pp. 280–285.

Preparations, Inventions, etc.

PEZPLANUS.

(T. Holland & Son, 46, South Audley Street, W.)

The Improved Instep Arch-sock for the cure of flat foot, patented by Messrs T. Holland and Son, is well known, and has been used with much success. As often happens in such cases, practical and continued experience has revealed the possibility of minor improvements which would tend to make the "Arch-sock" still more effective. Such improvements have been embodied in the "Pezplanus," recently contrived and patented by Messrs Holland & Son, and are as follows:

The "Pezplanus" has *two* transverse springs, one *under* and one *over* the longitudinal springs, instead of only *one* transverse spring, resting *on* the longitudinal springs as in the "Improved Sock." The longitudinal springs overlap each other in the centre of the arch, and thus allow of a slight movement which prevents fracture. The springs are firmly fastened together in such a manner as to impart greater strength, but at the same time allowing perfect elasticity. A more enduring support is thus secured, and greater comfort and adaptability ensured.

In the "Pezplanus," the leather moulded sole is of a much superior quality than that in the "Improved," and on this account will offer much greater resistance to dampness and perspiration. While retaining all the qualities for which the original supports were valued, there is no doubt that the "Pezplanus" will yield even better results in those severe cases, for which stronger and more effective support is needed.

THE "IDEAL" MEDICAL DIARY AND VISITING LIST.

(Langley & Sons, George Street, London, N.W.)

This Diary and Visiting List is in a form suggested by a member of the profession, and is an exceedingly comprehensive and convenient visiting "companion." The Diary is so arranged as to present, at an opening, space for sixty patients during each week, and it also contains suitable pages for registering obstetric engagements, nurses' addresses, patients' addresses, and other memoranda. There are no advertisements, nor any extraneous matter, so that the size and weight are not likely to cause inconvenience. A special feature is the addition of a Duplicate Refill Prescription Book, indeed this and the Diary and List may be obtained as Refills in a specially durable Morocco wallet. Prices: 7s. 6d. and 5s. 6d.

TABLOIDS AND SOLOIDS.

(Burroughs Wellcome & Co., London, E.C.)

Tabloid "Xaxa" and Dover's Powder.—As our readers are aware,[1] Tabloid "Xaxa" presents pure acetyl-salicylic acid, a valuable anti-rheumatic, anti-pyretic, antiseptic, and analgesic. In combination with Dover's Powder, it is indicated in influenza, catarrh, and various febrile and infective conditions, and its effect is to reduce temperature, destroy infection, and relieve pain.

The tabloids are issued in bottles containing 25 and 100, and the dose is from one to four as may be prescribed.

"Soloid" Eosin-Azur, for Giemsa Staining with one Solution.—Recent researches by Schaudinn, Metchnikoff, and others, point to the Spirochaete pallida as the probable causal organism of syphilis, and the identification of this organism largely depends upon the suitability of the staining reagents employed. Giemsa's method, which is recommended, requires a mixture of aqueous solutions of eosin, and of pure methylene azur. The use of "Soloid" Eosin-Azur enables the process to be carried out easily and conveniently with one solution.

Full directions for the use of this microscopic stain are issued with the soloids, which are packed in minute tubes of 6.

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[1] THE PRACTITIONER, Vol. LXXVII., p. 143.

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THE PRACTITIONER.

APRIL, 1907.

THE TREATMENT OF POST-PARTUM HÆMORRHAGE.

By G. ERNEST HERMAN, M.B., F.R.C.P., F.R.C.S.,
Consulting Obstetric Physician to the London Hospital.

THE EDITOR of THE PRACTITIONER has done me the honour of asking me to criticise three articles on the above subject, which appeared in the issues of August and November 1906, and February 1907.

The first of them is by Mr. Stanmore Bishop. He speaks of "a consensus of opinion backed by recognised authority," which he thinks needs to be upset. He wishes to change the teaching that, in order to check post-partum hæmorrhage, the uterus must either be made to contract, or "some astringent preparation, such as perchloride of iron, must be applied to its interior." I know not where this teaching is given. I thought the treatment of post-partum hæmorrhage by injection of perchloride of iron was now extinct. If it is not, I gladly join hands with Mr. Bishop in helping to do away with it: because (1) it cannot be relied upon to stop bad hæmorrhage, and (2) it leaves the uterus filled with dead clotted blood—an excellent soil for the multiplication of microbes—and therefore gives the patient an extra chance of blood-poisoning. Further on Mr. Bishop says that the student is taught that there are four kinds of post-partum hæmorrhage: that "his first duty is to satisfy himself under which head the particular case he has to treat will fall": and while he is making up his mind the bleeding is exhausting the patient. Again, I know not where such preposterously unpractical teaching is given. Mr. Bishop draws a verbal picture of a man called to a case of post-partum hæmorrhage whose first proceeding is to search for tears of the cervix, uterus, or vagina, presumably with speculum, a light, and swabs. Such a man, if he exists, ought scarcely to be at large: he is dangerous. A student, who should give that as his treatment of post-partum hæmorrhage, ought, without hesitation, to be referred. But I cannot imagine what teacher would tell him such stuff.

In another way, Mr. Bishop, though he correctly represents what is taught as to practice, yet fails, I think, to grasp its meaning. He says that the student is taught that, if in the intervals between uterine contractions, "he has been so impatient of the delay as to attempt by squeezing or otherwise stimulating the uterus before it was ready to contract once more, he may have done great harm by exciting irregular, spasmodic, and inefficient action." What "inefficient" uterine action in the third stage of labour is, I know not. Uterine contraction may take place or not; but if there is uterine contraction in the third stage of labour, it is always efficient. Most teachers, so far as I know, tell their students not to hurry the third stage of labour. After Credé had published his papers on expression of the placenta, he had enthusiastic followers, who were anxious to improve on the teaching of their master, and who therefore strove to squeeze out the placenta as quickly as they possibly could: and it is a fact that such hurry in squeezing out the placenta was followed by an excessive proportion of secondary post-partum hæmorrhage. But this was not because there was too much "excessive or spasmodic" uterine contraction, but because pressing out the placenta too soon means that the placenta pulls after it the membranes before they have been completely detached: and if a bit of chorion has not been completely detached, it is likely to be torn off and left in the uterus.

Mr. Bishop then goes on to enumerate 13 possible causes of post-partum hæmorrhage, and says that "all these should be present in the practitioner's mind in a sudden emergency like this." I know not whether he is expressing his own opinion, or trying to depict what he thinks students are taught. Some of his 13 causes the student may certainly put out of his head. "Too rapid succession of pregnancies" may be bad, but has never been shown to have anything to do with post-partum hæmorrhage. "Precipitate labour" is a guarantee against hæmorrhage rather than a cause of it. I know of no kind of "constitutional weakness or exhaustion" so definite that, when it is present, post-partum hæmorrhage may be anticipated.

And I know of no evidence that either drugs or diet during pregnancy have any effect upon the amount of bleeding during the third stage of labour.

These, however, are minor things. In one great point I must absolutely differ from Mr. Stanmore Bishop. I hold the current teaching, that it is uterine contraction which stops post-partum hæmorrhage, to be correct. The only effective treatment of post-partum hæmorrhage is to bring about uterine contraction. Pressure is only a temporary means of suspending bleeding until the uterus has regained its contractile power.

There are few topics more productive of verbosity than attempts to define "retraction" and "contraction" of the uterus. Dr. Hastings Tweedy refers to an author whose name he has forgotten, but who wrote four years ago. May I suggest that he, and others who are interested in the subject, should go back a little further and read the paper on "Elasticity, Retraction and Polarity of the Uterus," by Dr. Matthews Duncan, in the *Transactions of the Obstetrical Society of London*, Vol. XXVIII. (1886), p. 115? Of all men who ever lived Matthews Duncan was at once the most learned in obstetrical literature, and the most precise in the use of language; and any one who aspires to improve obstetrical nomenclature would do well to read Duncan's works first. I will not discuss definitions; but I will describe. Any one who puts his hand on the abdomen of a woman who has just been naturally delivered will feel the uterus. It is firm, its shape is definite; its inner wall is applied to the placenta; its muscular fibres are grasping, and constricting the vessels; and in this condition it remains. This is *retraction*. Every few minutes the uterus becomes smaller and harder; it grips the vessels more tightly, and it squeezes the placenta; and if the placenta is loose it squeezes it out. This is *contraction*. The patient is not safe in the third stage of labour until this condition of *retraction*, with intermittent *contraction*, has come to stay.

It is well known that in every place, where there are a number of medical men, there are some who frequently have to treat post-partum hæmorrhage, and others who never see it. There are some who, if the progress of labour is slow, think it wrong to wait, and therefore seize the child either with forceps or by its foot, and pull till it comes. These are the men who often have to treat post-partum hæmorrhage. There are others who watch uterine action, and avoid pulling when the uterus is not contracting: these men never see dangerous hæmorrhage. The great prophylactic against post-partum hæmorrhage is to avoid emptying the uterus when it is not contracting. Forceps, or foot traction, should be used only to *help* uterine action, *never to replace* it. I am glad to see the Master of the Rotunda preach this sound doctrine. I learned it from Braxton Hicks; it is, as Hicks described it, the "very keystone of good midwifery." Mr. Bishop speaks of "sesquipedalian Latin, Greek or German terms" (I wonder where he has heard them), which are apparently somebody's denomination of "atony of the uterus" or uterine inertia—terms which Mr. Bishop would replace by the word a "tired" uterus. A tired uterus has been described by Braxton Hicks as a uterus in a state of "temporary passiveness," a very good descriptive term. By Scanzoni the condition was called "secondary uterine inertia." Mr. Bishop applies to it a word which is shorter, and I offer no objection to his doing so. But a tired uterus ought not to cause post-partum hæmorrhage, because a tired uterus will not expel the child, nor can it cause any damage to the maternal soft parts, and the accoucheur ought on no account to extract the child from a tired uterus. The cure for a tired uterus is the same as that for fatigue of any part of the body: sleep. If the accoucheur will give his patient some opium to make her sleep, when she wakes uterine action will return, the child and placenta will be expelled, and hæmorrhage in the third stage of labour will not be enough to cause anxiety.

A "tired" condition of the uterus is not the only cause of post-partum hæmorrhage. In the practice of good accoucheurs, it is sometimes provoked by the retention of a succenturiate placenta, or a bit of chorion in the uterus. If hæmorrhage is thus caused, the proper treatment is to remove the cause of the hæmorrhage, and, when this is done, the hæmorrhage can be stopped.

When Emmett introduced his little operation of sewing up tears in the cervix uteri, his numerous and devoted friends and pupils vied with one another in magnifying the benefits to be obtained from this trivial operation; and, therefore, magnifying also the pathological importance of tearing of the cervix. Among the alleged consequences of tears in the cervix figures post-partum hæmorrhage. One cannot say it is impossible for a torn cervix to cause dangerous post-partum hæmorrhage, but I doubt if it ever does. I have never seen considerable hæmorrhage from this cause, nor met any one who had. Rupture of the uterus is often fatal, but it does not lead to death so quickly as hæmorrhage. When an artery is torn across, its muscular coat has a trick of contracting so as to close the vessel. Therefore, I think that fatal hæmorrhage from a torn cervix has occurred only in the imagination of persons loyally and honourably anxious to magnify the work of Dr. T. A. Emmett.

Mr. Stanmore Bishop's remedy is pressure. He draws another vivid word picture of a surgeon, who, without artery, forceps, or ligatures, is called to a man whose leg has been taken off by a circular saw, and who squirts hot water at the stump, tries to compress it with gauze, and paints it with perchloride of iron. The suggestion is that he should elevate the limb and compress the abdominal aorta. I think most surgeons would put a tight bandage or ligature round the limb, and and thus compress the vessels until forceps and ligatures arrived.

I agree with Mr. Bishop in the opinion that, when with dangerous post-partum hæmorrhage uterine contraction is absent, pressure is the best thing. But I think pressure is better applied, not to the abdominal aorta, but to the uterus. Bimanual compression of the bleeding points is, in my judgment, the best way of stopping post-partum hæmorrhage. The body of the uterus is the part that should be compressed. To bend the neck of the womb, by pressing the cervix forward, as some have recommended, seems to me bad practice, for it blocks up the cervical canal, and thus prevents any clots from being squeezed out.

The curious thing is that Mr. Bishop should write as if he thought compression of the aorta in post-partum hæmorrhage was something new. It was extolled by Baudelocque the younger before the French Académie des Sciences in 1835, and F. H. Ramsbotham states that he had employed the practice long before Baudelocque's memoir had been published. Ramsbotham says: "There is not much difficulty in controlling the flow of blood through this vessel in most women after delivery, especially if they be of spare habit, owing to the lax state of the abdominal muscles; and in some cases of after hæmorrhage this proceeding may be attended with fortunate results."[1] Robert Barnes[2] gives a list of authors, who have recommended it, and says that he himself considers it "a momentary resource." Most text-books since have mentioned it.

Post-partum hæmorrhage is a terribly acute danger; and its treatment must be prompt. The first thing in treatment is to put the right hand on the abdomen, to knead and grasp the uterus so as to make it contract. This can be done immediately; no preparations are required. Mr. Bishop postulates a uterus which is so tired that it will not contract. Then the next thing should be to put the left hand in the vagina, and compress the body of the uterus between the left hand with the fingers folded in the palm in the vagina, and the right hand on the abdomen. In this way the bleeding vessels can be compressed, and the uterus is at the same time stimulated to contract. In most cases bleeding will be stopped by pressure, and uterine contraction will follow. If it should not, or if the contraction excited is followed by relaxation, the accoucheur should remember that there may be something in the uterus which is preventing it from contracting; and therefore he should put his hand into the uterus to feel if there is a bit of placenta, or membrane, or a fibroid, which is preventing the uterus from contracting, and, if there is any such thing in the uterus, he should remove it. The insertion of the hand into the uterus will stimulate that organ, and generally make it contract, but contraction cannot be complete while the surgeon's hand is in the uterus. We shall all agree with Mr. Bishop in horror at the idea of squirting hot water at a divided limb to check bleeding, but the uterus is not like a divided limb. Squirting hot water into the uterus will often make it contract; and it will wash the inside of the uterus, and wash out clots and fragments of membrane small enough to escape the fingers of the accoucheur. Therefore, it is a good thing for the accoucheur, after he has put his hand in the uterus and made sure that there is nothing retained, to inject hot water. But all these things are only helps to the great object of getting the uterus permanently contracted and retracted. If the contraction induced by injecting hot water does not last, then I agree with Mr. Bishop in trusting to pressure, but pressure on the bleeding part, not on the aorta. Compression of the uterus will not only stop the bleeding, but it will directly provoke uterine contraction, which pressure on the aorta does not. I have never tried how long I could compress the aorta, and therefore I accept Mr. Bishop's statement that it can be compressed (so that no blood goes to the lower half of the body) for three hours. I do not think it would be necessary to compress the uterus as long as this before uterine contraction came on. Mr. Bishop holds it important to prop up the foot of the bed. This will do no harm, but it takes a little time which is better spent in making the uterus contract.

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[1] *Obstetric Medicine and Surgery*, 5th Ed., 1867, p. 464.

[2] *Obstetric Operations*, p. 462.

Mr. Bishop objects to attempts being made to replace an inverted uterus, or to remove an adherent or retained placenta, on the ground that "any interference of this sort is almost certain to be done with unsterilized hands," for the surgeon cannot wait to properly purify his hands. This objection ought only to apply to a case in which the surgeon happens to knock at the street door at the very moment when furious hæmorrhage is threatening life. In such a coincidence I grant that the temptation would be great for the surgeon to forget that his hands might be dirty. But if the surgeon has been in attendance during the second stage of labour, he will have made his hands aseptic before he touched the patient ; and he will have ready at the bedside basins of germicidal solutions in which to immerse his hands whenever soiled. In bleeding from inversion of the uterus, I think the proper treatment is at once to reduce the inversion ; just as in bleeding caused by a retained placenta, the thing is to remove it.

Dr. Gordon Fitzgerald describes an ingenious manœuvre, which is new to me, for ascertaining whether the placenta has been expelled into the vagina, viz., drawing the cord out of the vagina, then observing the effect upon it of lifting up the uterus by the hands on the abdomen.

I agree with Mr. Stanmore Bishop in his implied disapproval of plugging the uterus for post-partum hæmorrhage. This practice has, in my opinion, been discredited by its own advocates. When we read that one man has found it necessary many times in a single year to plug the uterus for post-partum hæmorrhage, the inference is irresistible that in most of them the plugging was unnecessary; and when we further find reports of cases of post-partum hæmorrhage which died in spite of plugging, the general conclusion follows that, in dangerous post-partum hæmorrhage, plugging cannot be relied upon.

In his concluding article, Mr. Bishop again attacks "the men who advise, at such a time, that a series of experiments should be made." I do not know who the men are who recommend "a series of experiments" in cases of post-partum hæmorrhage. The measures which I have described are not experiments, but manipulations, each with a definite and necessary object, the final aim being to secure that the uterus shall be empty, clean, contracting, and retracted in the intervals between the contractions. If contraction cannot be got, I agree with Mr. Bishop in trusting to pressure; but I think pressure is better applied to the bleeding part, viz., the uterine body, than to the abdominal aorta. I may point out, however, that it is possible to do both: for the doctor to compress the uterus, and the nurse the aorta, or *vice versâ*. Lastly, the great prophylactic is *not to deliver when the uterus is tired.*

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THE MYOGENIC THEORY.

By JOHN COWAN, M.D., (Cantab.), D.Sc., (Glasgow),
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The current theory of the cardiac action is in some respects a revival of an old idea. Haller had maintained that the beat of the heart was the result of impulses which originated in the muscle itself, but the discovery of nerve cells within its substance, and the command of the nervous system in the case of the skeletal musculature, led to the abandonment of this view. Dr. Gaskell's work on the subject, however, showed that Haller's theory was correct, and that the influence of the nervous system on the heart was in no way comparable with its influence on the voluntary muscles. The isolated apex of the ventricles, which does not contain any nerve cells, may, for example, in favourable circumstances, maintain a rhythmical beat for a considerable time, and the fœtal heart beats regularly at a period when no nervous connections with it exist.

The rhythmical contraction of the cardiac muscle is, then, the result of its inherent power to produce rhythmical stimuli, but we know nothing of their nature. These stimuli, in normal circumstances, originate a contraction at the venous ostia, which passes downwards through the auricles to the ventricles. All the cardiac muscle possesses the power of originating stimuli, and a contraction, set up in the ventricles, will pass backwards through auricles to venæ cavæ. The normal process of events seems to be due to the more frequent production of stimuli at the venous ostia.

The wave of contraction passes over the heart at a variable rate, as the conductive power of the several parts differs, the result, according to Dr. Gaskell, of morphological differences in the tissues, and this is probably beneficial, inasmuch as it tends, in unfavourable conditions, to produce a more regular emptying of the ventricles than would occur, if the conductive power was everywhere equal (Engelmann). Dr. Gaskell, too, showed conclusively that the stimulus is conducted by the muscle and not by the nerves. The connecting nerves in the tortoise, for example, can be completely severed without interfering with the regular sequence of events, while section of the muscle alone is followed by asynchronous contractions of the separated parts.

His experiments were at first alleged to be inconclusive, and not applicable to mammalian hearts, as no muscular connection between their auricles and ventricles was at that time known, but Dr. Kent and His jun. shortly afterwards demonstrated the connection, and Tawara, and Drs. Keith and Flack have recently worked the point out. The bundle of His, or the auriculo-ventricular bundle, takes origin in the annular fibres of the right auricle, and perforates the central fibrous body of the heart, which lies between the auriculo-ventricular fibrous rings and is the main connection between them, the aorta, and the base of the ventricles. It then passes downwards and forwards along the lower margin of the membranous septum, and, dividing, is distributed to the two ventricles on each side of the ventricular septum. Its relations to the membranous septum and the mitral and tricuspid valves are important.

The cardiac muscle differs in other respects from skeletal muscle. In the ordinary muscle-nerve preparation the degree of reaction to a stimulus varies with its strength, but the cardiac muscle, if stimulated, responds with the maximal contraction of which it is capable at the time. Repeated stimuli, too, never produce tetanus, for each contraction of the muscle is accompanied by a "refractory period," during which no reaction can be produced by stimulation. The refractory period commences shortly before the contraction occurs, and lasts until a short time after its termination, and the excitability is only gradually restored, reaching its maximum just before the occurrence of the next normal systole. Engelmann has examined this refractory period in some detail by the application of isolated electrical stimuli at varying periods of the cardiac cycle.

DISTURBANCES OF EXCITABILITY.

The Extra-systole (*Premature Systole*).—Experimentally, an extra-systole can be easily produced by mechanical, thermal, chemical, or electrical stimulation. If an isolated electrical stimulus is applied to the heart, during its excitable period (*i.e.*, during diastole), an isolated contraction follows, and the pulse wave is larger the longer the interval between the stimulus and the preceding normal beat. The contractility of the heart, absent altogether during the refractory period, only gradually recovers during diastole. If the rhythm of the heart is regular, the succeeding physiological stimulus from the venous ostia reaches the auricles during their refractory period, and no cardiac contraction in consequence obtains until the next (third) physiological stimulus is applied. An extra-systole is thus followed by a pause of longer duration than normal, and of such a length that the interval between the two physiological beats is exactly double that of the normal rate (Fig. 1). The third (post-compensatory) pulse-wave is always larger and stronger than normal, for the longer diastole induces an increased contractility, and more blood of necessity will enter the ventricles during the longer pause.

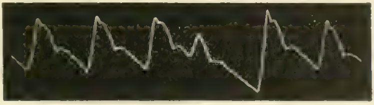

Fig. 1. *The fourth pulse wave (extra-systole) occurs at an earlier period of diastole than the average. The fifth wave is later as well as larger, and the interval between it and the third is exactly double the average period.* (Dr. James Mackenzie.)

An extra-systole may be produced in the frog by stimulation of either auricle or ventricle ; in the mammalian heart, however, a full compensatory pause does not invariably follow auricular stimulation, the diastole being sometimes too short ; on ventricular stimulation, the full pause always follows. No compensatory pause follows stimulation of the frog's sinus, or the mammalian venous ostia ; the extra-systole is followed by a physiological beat after a diastole of normal length ; and the shortening of the pause, after auricular stimulation in the mammal, may perhaps be explained by the fact that, in the mammalian development, a portion of the sinus goes to form the auricle.

Extra-systoles, with full compensatory pauses, are not uncommonly met with in clinical work. They may be observed in sphygmographic tracings, or even on digital examination. They may, too, produce an intermission instead of an irregular beat, for, if they occur early in diastole, the result of the contraction is small, as the ventricle is as yet imperfectly filled and its contractility is but slight, and the aortic pressure may be still so high that no result of the imperfect systole is recorded in the radial pulse. An intermission of the radial pulse may, then, be simply an expression of an irregular stimulus occurring at a time when no radial beat is possible; and in consequence, as Dr. Mackenzie has pointed out, there is no real difference between an irregularity and an intermission. An intermittent pulse is not necessarily a greater danger than an irregular one.

Extra-systoles are less commonly present in rapid pulses than in slow ones, and they frequently disappear if the rate of the cardiac action is increased by alterations in position, exercise, etc. The reason for this is not difficult to appreciate. With more frequent beats, the refractory period in a given number of seconds is largely increased, and the opportunity for extra-stimuli to be effective, is thus considerably lessened.

"It is a remarkable fact that the regular automatic rhythm of the heart is not affected by the ordinary extra-systoles that proceed from the auricle and the ventricle."[1] The rhythm is unchanged or, at any rate, the rate of stimulus production. An abnormal factor is introduced with an abnormal occurrence, but this does not predicate an abnormal condition of the heart. All the trend of evidence in the meantime, however, shows that the stimulus must be applied directly to the cardiac muscle.

If further investigations show that this is always the case, an extra-systole must be regarded as evidence of some abnormal stimulus to either auricles or ventricles; but the stimulus may be of very varied nature. It must also be remembered that nervous influences, though they do not originate or conduct stimuli, may apparently affect these muscular properties in either a negative or a positive way, and the exaltation or depression of these functions may allow of results from causes which would not be active in a normal condition of affairs.

[1] Wenckebach, p. 47.

Dr. Mackenzie's tracings show that extra-systoles may be due to both auricular and ventricular stimuli. In his book he shows numerous tracings where the jugular and radial pulses show correspondence, as well as others in which the ventricle alone participates. In the accompanying tracing (Fig. 2), an extra-systole is seen to occur at ×: c' in the jugular tracing corresponds in time with ×, and is therefore the carotid expression of the extra-systole, but it is succeeded by an auricular pulsation a', which occurs at the moment when the normal auricular beat should obtain, *i.e.*, the auricular rhythm is unchanged, although an extra-systole of ventricular origin has been interpolated. The radial irregularity in the two cases is precisely similar; examination of the jugular pulse, however, reveals the essential difference.

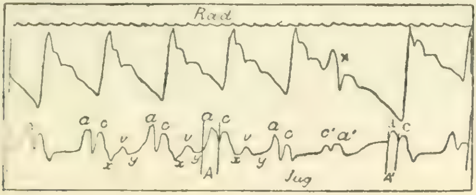

Fig. 2. (Dr. James Mackenzie.)

Dr. Mackenzie classifies irregular pulses in two main groups : the youthful type, in which the duration of the systole is unaltered, while that of the diastole varies within considerable limits ; and the adult type, in which the systole and the diastole may both vary in length, many of these cases being the result of extra-systoles. Wenckebach agrees with Dr. Mackenzie that extra-systoles are uncommon in childhood, but he can draw no special conclusion with regard to their occurrence in adults. He has found them in persons in perfect health ; in those who are suffering from cardiac and circulatory troubles ; and in those who are affected with diseases which have no direct cardiac association.

DISTURBANCES OF CONDUCTION.

Disturbances of Conduction.—The phenomena of "Gaskell's bridge" are of course classical and well known, and their clinical correlation is important. The original experiments were carried out on the heart of the tortoise. Dr. Gaskell found that, if he made an incision through most of the auricular walls, so that only a narrow strip of tissue connected auricles and ventricles, a delay occurred in the passage of the wave of contraction over the bridge ; and its further narrowing produced so great a delay that ventricular contraction only succeeded every second, third or fourth auricular pulsation. A "heart-block" was established.

An abnormally slow pulse is a not uncommon occurrence in clinical work, and a true and a false bradycardia have been described. In the first group, the radial pulsations correspond with the cardiac beats, as recognised on auscultation. In the second, some of the cardiac beats are so feeble that they convey no appreciable pulsation to the radial artery. The sphygmograph shows that, in many of these cases, a radial pulse is really present, though the movements are too small to be recognised by the finger, but, in a certain number of cases, no radial pulse accompanies the cardiac beat ; ventricular contraction, if it occurs, is so feeble that the semilunar cusps are not raised. Dr. Mackenzie's methods of investigation have opened out a new field of observation. He pointed out that the radial pulse was the result of the contraction of the left ventricle, and that its observation conveyed no indication of the state of affairs on the right side of the heart. In many cases, however, pulsation might be noticed in the jugular veins, in the liver, the epigastrium, etc., and he has devised clinical methods for the registration of these pulses simultaneously with that of the arteries.

The venous pulse is, of course, expressive of the blood pressure in the right auricle, and, in some of these cases of slow pulse, tracings show that the auricular pulsations are more numerous than those of the left ventricle, as gauged by the radial pulse. A condition exactly similar to Dr. Gaskell's heart-block obtains. (Fig. 3.)

Heart-block may be either partial or complete. Experimentally a 2-1, 3-1 rhythm can easily be produced, and, if the path between auricles and ventricles is still further interfered with, the ventricle finally ceases to beat, although the auricular rhythm remains unchanged. After a short period, however, the ventricle again contracts, but its rhythm is its own and quite independent of that of the auricle. In Fig. 4, the auricular and ventricular beats, although both are rhythmical, are seen to occur independently of each other.

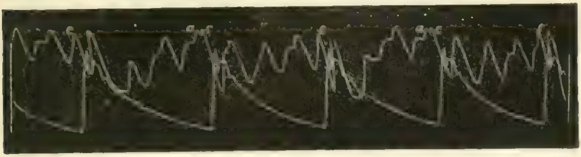

Fig. 3. *Tracings from the jugular vein* (*upper curve*) *and radial artery* (*lower curve*), *showing nervous pulsations in the interval between the radial beats.* (Dr. G. A. Gibson.)

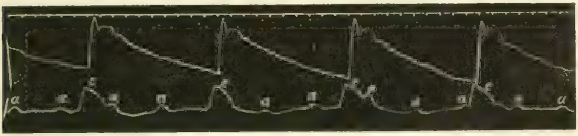

Fig. 4. *Shows the complete dissociation of the auricular waves* (*a*) *from the carotid pulse* (*c*). *The fifth auricular wave is hidden by the simultaneous occurrence of the second carotid pulsation.* (Dr. James Mackenzie.)

Reference has already been made to the situation of the auriculo-ventricular bundle. Its cells (Purkinje's fibres) differ in many respects from the ordinary cardiac muscle cells. They are much larger and of paler colour ; they are fusiform in shape, and possess a fibrous sheath ; the central mass is undifferentiated, and the periphery of the cell alone is striated. The difference in structure indicates a different function, and the conducting properties of the part are still further suggested by the distribution of the bundle, and by the fact that its bulk is not affected by causes which produce atrophy or hypertrophy of the whole heart (Keith, Aschoff). His jun., in 1899, suggested that the bundle was the bridge over which the cardiac contraction passed, and Erlanger, in 1905, proved that his suggestion was correct, as he was able to produce a heart-block in dogs by its compression *in situ*. Dr. Keith has recently advocated caution in the acceptance of the theory that the *a.v.* bundle simply subserves the function of conduction. In Dr. Chapman's case the ventricular portion of the bundle was intact, although its upper part was wholly destroyed. And as abrogation of function is almost invariably followed by atrophy of the part, it seems probable that the bundle is something more than a mere connecting link for the transmission of stimuli.

The Stokes-Adams syndrome seems to be intimately associated with heart-block. The cerebral attacks, whether syncopal, apoplectic, or epileptiform, are apparently always associated with a paroxysmal diminution in the number of the radial pulsations, and the pulse may be entirely absent for many seconds, the longest interval hitherto recorded being 2 minutes 10 seconds (Stengel). It was suggested by Tripier that the cerebral symptoms were the essential feature of the attacks, and that the cardiac disorder was the sequel of the cerebral, or simply an accidental coincidence ; but, from Holberton onwards, observers have recorded that the pulse failed before the cerebral seizure, and that a return of consciousness coincided with the return of the pulse (*cf.* A. Webster's tracings). Huchard seems to consider that Tripier's views are correct, and his classification of the syndrome as "Artério-sclérose du cœur : forme cardio-bulbaire" indicates in a fairly definite manner his view of its causation. Dr. Gibson, too, considers that the onset of the nervous symptoms is due to sclerosis of the cerebral vessels. In such cases, "whenever there is any stress put upon the circulation, the blood supply to the brain is inadequate and ineffective. There is, as a result of that deficient supply, a condition of irritable weakness of the inhibitory mechanism ; impulses are sent down to the heart ; it is inhibited, and the features of the attack are found."[1]

It is perfectly true that arterio-sclerosis is a common feature in patients who suffer from these symptoms, but, in Dr. Hay's case, at any rate, the vessels were normal, and, in Medea's case, though the circle of Willis was degenerate, the nutrient vessels of the bulb only showed infinitesimal alterations. And it seems possible that the whole of the symptoms may be due to the ineffective cardiac action. As Dr. Gibson points out, some special stress usually, though not invariably, precedes an attack, and it is known that more frequent, or more forcible, action of the heart tends to diminish the conduction power. And, until this function is restored, the blood supply to the brain must be inadequate, and, as Kussmaul and Tenner have shown experimentally, epileptiform seizures may result from cerebral anæmia. There seems to be no necessity for the introduction of cerebral inhibition ; the stress itself may produce a prolonged defect in the cardiac action, but it must be acknowledged that grave cerebral symptoms are more likely to occur, if the cerebral arteries are degenerate and unable to react satisfactorily to abnormal conditions. Erlanger has adduced a cogent argument against Dr. Gibson's views. Inhibition of the ventricles would necessitate inhibition of the auricles as well, and all his venous tracings show that the auricular rhythm is not slowed during attacks.

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[1] G. A Gibson : *Morison Lectures*, 1904, p. 55.

Stokes, in his paper, recorded the presence of jugular pulsations and cardiac sounds, which were not represented in the radial pulse, so that it seems clear that his case was an example of heart-block. It is impossible to consider here all the cases of heart-block which have now been published, and I shall only refer to those in which the evidence of interference with the auriculo-ventricular bundle is more or less complete.

Sendler, in 1892, described a case in which a cartilaginous tumour, the size of a walnut, was situated in the interventricular septum about a centimetre beneath the ostium of the pulmonary artery. Dr. Handford, in 1904, published the records of a case of heart-block, in which post-mortem examination revealed the presence in the interventricular septum of several gummata, which must, almost certainly, have involved the auriculo-ventricular bundle. In Dr. Hay's first case, the auricular canal was stretched, and the junction between the base of the tricuspid valve and the base of the ventricle was stretched and attenuated, which would necessarily involve the auriculo-ventricular bundle. In his second case, the artery of the central fibrous body was atheromatous, and the bundle at this point was involved in an inflammatory mass. In Stengel's case, an atheromatous patch at the base of the anterior mitral leaflet extended into the septum and involved the bundle of His. In Schmoll's case, the bundle was involved in a fibrotic mass. In one of Aschoff's cases, examination showed severe sclerosis of the small arteries, which traversed the central fibrous body and the principal trunk of the conducting bundle; in the other, these parts were greatly infiltrated with fat. In Dr. Gibson's case, the bundle was scarred by fibrous tissue, and its cells were atypical. Sir James Barr's case is reported in fuller detail than any yet published. His tracings of the jugular and radial pulsations show clearly that auricular contraction took place during the ventricular pauses, and that the convulsive attacks succeeded the ventricular failure. The longest radial pause lasted for 90 seconds. Dr. Keith reported that the central fibrous body was atheromatous, and the auriculo-ventricular bundle fibroid. (Sir James advocates caution in the acceptance of the myogenic theory, and does not consider that the current theories are as yet proven.) In Dr. Chapman's case, the commencement and the upper half of the main *a.v.* bundle were completely destroyed by their involvement in a diffuse fibrosis, which was probably of syphilitic origin. In the case reported by Vaquez and Esmein, the bundle was involved in a gummatous mass which was situated in a lower part of the interauricular and the upper part of the interventricular septa. In the case reported by Jellick, Cooper, and Ophuls, the bundle was involved in an infarct of the muscular septum.

It is not difficult to understand how the bundle is affected. Its situation, in close proximity to the valves and the undefended space, must render it peculiarly vulnerable, for, as is well known, endocarditis is always associated with more or less damage to the myocardium, and the undefended space is generally large in high-tension cases. The musculi papillares are supplied with Purkinje fibres, and are usually notably fibroid in old standing cases of mitral disease. Even, without a full heart-block, disturbances of rhythm may be due to interference with some of the paths of conduction.

It must not, however, be supposed that every case, in which the conduction is interfered with, has a gross lesion in the auriculo-ventricular bundle. The conduction of stimuli, like their production, is influenced by the cardiac systole, and each beat abolishes, or lessens, the function to a notable extent, though it is gradually restored during diastole; the stronger and more numerous the contractions the greater the result.

Experimentally, defects in conduction are found to occur, if the heart is in a state of asphyxia, and the administration of oxygen in these cases improves the conduction power; while, if it is defective, the defect is intensified by the administration of digitalis (Mackenzie).

At the present time, we have very little accurate knowledge of the pathological conditions obtaining in cases of heart-block, as the position and nature of the auriculo-ventricular bundle have only recently been recognised; but all the recorded cases have showed evidence of damage at this place. Holberton's case, however, which has been so often quoted, seems possibly not to have been an example of this lesion. In this patient, a fall in the hunting field was followed, after an interval of two years, by the Stokes-Adams symptoms. The accident was associated at the time with symptoms of injury to the cervical vertebræ, and post-mortem examination showed that the foramen magnum was narrowed, and the medulla and upper cervical cord compressed. The nervous system has rarely been examined with modern methods in this disease, and, in the two cases, which I have found recorded, the bulbar nuclei and roots were apparently normal.[1]

Dr. Gaskell and His jun., however, have shown experimentally that heart-block can be produced by vagus stimulation, and Dr. Mackenzie has published examples of heart-block due to swallowing, acting presumably through this nerve. Further experience may show that nervous influences are more important than is at present recognised.

It must also be remembered that heart-block is not always accompanied by the Stokes-Adams symptoms. In some cases the conductivity may be deficient, or even apparently wholly absent, and the auricular and ventricular contractions may be asynchronous without the occurrence of cerebral symptoms. The auricular rhythm, in such cases, may be regular (Fig. 4), or irregular (Fig. 3), but so long as the ventricle is capable of supplying sufficient blood to the brain, no such seizures occur. All the cases of the Stokes-Adams syndrome, which have been fully reported, have shown a *prolonged* paroxysmal failure of ventricular contraction, thus differing completely from the tracings shown. In Dr. Mackenzie's case, the pulse abnormalities were known to exist for more than two years. Dr. Gibson's patient was dying as the result of pericarditis, at the time when the tracings were taken.

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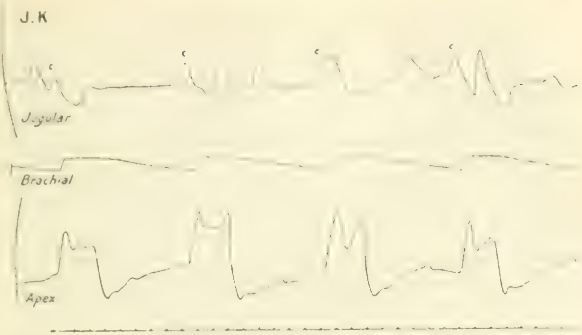
[1] Medea reports two cases, but the second case is recorded from the clinical side in an incomplete manner, and seems probably not to have been an example of the Stoke-Adams syndrome. The cardiac examination in both cases was incomplete, and the auriculo-ventricular bundle was not examined. In Brouardel's case, the cerebral arteries were degenerate, but not "in sufficient degree to justify a bulbar origin for the attacks"; the heart was not examined in detail.

Defects in conductivity are, moreover, of importance from the therapeutic side. The administration of atropine, for example, has been shown (Ritchie, Gibson) to increase the frequency of the auricular beats in these cases, while the rhythm of the ventricles remained unchanged, and Dr. Mackenzie's evidence of the evil influence of digitalis is also of clinical value. The action of drugs upon the heart must thus be investigated afresh, and there is little doubt that fuller knowledge will lead to more accurate indications, or contra-indications, in favour of this or that drug.

Dr. Gibson has pointed out that heart-block may be due in some cases to defects in the excitability of the muscle, without any conduction fault. At the Toronto meeting of the British Medical Association, he showed tracings, and narrated the history of a case of heart-block which he believed was due to such a cause.

He has also drawn attention to two other methods by which heart-block can be recognised. The fluorescent screen can show the auricular and ventricular movements, and variation in their rhythm can easily be appreciated. His studies on the electro-motive changes, which accompany the movements of the heart, have recently been extended to cases of heart-block, and he has demonstrated in these cases waves, which are evidently due to auricular systoles, interpolated between the waves of ventricular origin.



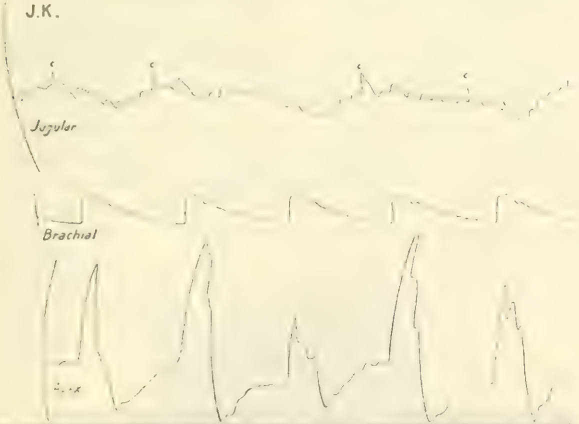


Fig. 5a. *This tracing shows absolute disassociation of auricular and ventricular action. There are irregular interferences of the auricular with the ventricular pulsations, and the carotid and auricular impulses show a very variable relationship. Some of the auricular pulsations are conducted to the tracing obtained from the apex, but do not appear on the brachial curve. The rate of the ventricular pulsation was 36. The auricular was difficult to count, but was usually about 90. This tracing shows complete heart-block.*

Fig. 5b. *From the same patient as that from whom Fig. 4 was obtained, after the administration of atropine. It shows that the drug produced its usual effect upon the auricular pulsation, without exerting any influence over the ventricles, the ventricular rate being 36 and the auricular 275. The tracing shows the conduction of some auricular impulses to the apical curve, but these cannot be observed in the brachial tracing.* (Dr. G. A. Gibson.)

DISTURBANCES OF CONTRACTILITY.

Disturbances of Contractility.—The contractility of the cardiac muscle can be easily gauged experimentally, for, as has been already mentioned, an effective stimulus always produces the maximal contraction of which the heart is, at the time being, capable, and the size of the contraction is thus a measure of the function. And this special property can be shown to vary on the administration of various substances, as well as under nervous influences. Wenckebach has brought forward evidence which tends to show that the pulsus alternans is the result of a diminished contractility of the cardiac muscle. It has been shown, by experiment, that this is a possible cause of such irregularities, and its origin has been explained as follows.

The contractility of the heart is destroyed by its systole, and the power of contraction is only gradually restored during diastole ; and the duration of the contraction is also affected, its length being shortened by diminution of the function. If then, from any cause, in a heart, in which contractility is deficient, the period between beats is but once lengthened, the contraction which follows will, from the increased contractility after the longer pause, be both larger in amount and longer in duration than those preceding it. The diastole, if the normal rhythm is maintained, will, in consequence of the longer beat, be shorter than normal, the restoration of the contractility will be incomplete, and a smaller and shorter systole will result. The longer diastole will allow the next beat to be again lengthened and strengthened, and the same results will recur indefinitely.

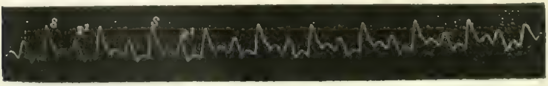


Fig. 6. (Dr. James Mackenzie.)

The pulsus alternans must not be confused with the more common bigeminal pulses which result from extra-stimuli. The distinction is, however, easy. Extra-systoles are always followed by a compensatory pause (Fig. 1). In the pulsus alternans (Fig. 6) the interval between the beats is practically equal. Isolated examples of the former are common; the pulsus alternans, on the other hand, continues unchanged for weeks or months.

The conditions, which induce this diminution, are, however, not well defined. Rapid action of the heart, asphyxia, and such lesions as pericarditis and myocardial fibrosis, must, however, exert a more or less great influence, and some of the drugs of the digitalis group have a similar action.

There is but little information available concerning the other properties of cardiac muscle, those of stimulus production and tonicity. Irregularity of stimulus production may presumably be a cause of an irregular pulse, but we know little of the normal mechanism, and still less of its abnormal working. Dr. Gaskell believes that the cardiac muscle, like the systemic, has a normal "tone," and has shown that it may be altered by the application of various drugs. It has been suggested that dilatation of the heart may be due to a loss of tone, but the evidence of this is, as yet, inconclusive.

Dr. Mackenzie's researches on the venous pulse have been already referred to; and we should have liked to consider some more points on which he has thrown considerable light; but we are only able to refer in detail to one other clinical point.

A tracing of liver pulsation, under the usual conditions in which it is present, shows two or more elevations (Fig. 7).

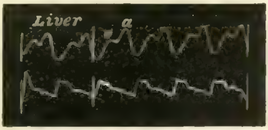


Fig. 7. *Tracings taken in* 1893. (Dr. James Mackenzie.)

The first and largest wave corresponds to the auricular contraction; the second and smaller is the expression of the right ventricular systole communicated through the incompetent tricuspid valve; and the greater the tricuspid incompetence, *the earlier and the larger* will the second wave be. In many cases of old standing mitral disease, the dilated auricles, as seen *post-mortem*, are evidently incapable of contraction, and, in cases of this description, the auricular wave may wholly disappear from the tracing, and the ventricular wave alone obtain (Fig. 8). This alteration in the character of the venous pulse,

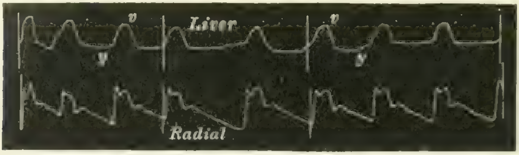


Fig. 8. *Taken from same case as* Fig. 7 *in* 1898. (Dr. James Mackenzie.)

is then indicative of right auricular failure, which may be either permanent or temporary, but which is at any rate evidence of the degree of failure of compensation. The prognosis, in cases of old-standing mitral disease accompanied by more or less muscle failure, is often very difficult, as the right heart is hidden by the lungs, and accurate observation is in consequence impossible ; but, by Dr. Mackenzie's ingenuity, another datum has been obtained, and one too which continued observations will probably show to be of great value.

The literature on the subject is already enormous, and only the more important papers are referred to below. Bibliographies will be found in the items marked *. I have great pleasure in acknowledging my debt to Dr. G. A. Gibson and Dr. James Mackenzie for the figures utilised in this review.

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SIX CASES OF RUPTURED ECTOPIC GESTATION.

FIVE EXSECTIONS—FOUR RECOVERIES.

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[With Plates X.—XI.]

ALTHOUGH the cases submitted to the reader in the following pages present no unusual features, the subject of ectopic gestation is one of such great importance, to practitioner and patient alike, that they furnish material of sufficient interest to record. The occurrence of ectopic gestation, in the experience of any individual general practitioner, must be an uncommon one, and, consequently, one that is liable to be overlooked, unless published cases serve the purpose of keeping the subject continually before the professional mind. Unlike some rare affections, its diagnosis is usually easy, and it is of such capital importance to the patient that it should be recognised, and recognised early, when it does occur, that a complete familiarity with its symptoms and signs should form part of the equipment of every practitioner of midwifery. Though, comparatively speaking, it is a rare event, yet the practitioner must be ever on the look out for it, since promptness in its recognition and treatment will, in all cases, succeed in rescuing his patient from a pressing danger, and, in many, from the very jaws of death itself. It is the early symptoms, before rupture, that require particular attention. After that catastrophe, any treatment may be "too late."

Case I.—F. R., æt. 31. Seen in consultation with Dr. A. Reid, of Southsea.

Previous History.—Always very regular, with scanty period, used to suffer with pains in the pelvis on exertion, one child, 11 years old; no subsequent pregnancy.

Present Illness.—She missed her period, due March 19, 1905, had morning sickness, and considered herself pregnant. A fortnight later, noticed a coffee-coloured vaginal discharge, which was present daily. On April 14, she was suddenly seized with severe crampy pain in the lower abdomen, accompanied with faintness, which lasted 2½ hours. No vomiting. During this attack, the vaginal discharge was bright red, but subsequently became dark again. On April 19, the discharge became bright again, and resembled an ordinary period. This continued till May 1, on which day she had another severe attack of cramp in the lower abdomen, accompanied with faintness and vomiting. Similar attacks occurred on May 2, and 3 on which day I saw her, in consultation with Dr. Reid.

Condition at Consultation.—Patient was confident, from her own feelings, that the missed period meant pregnancy. She complained of severe crampy pain in the lower abdomen, of faintness, and of great thirst. She was blanched. There were no changes in the breasts. The abdomen was uniformly distended and very tender all over. No dulness was present, but the right loin was flat in note as compared with the left. Liver dulness present. Urine drawn off ℥xx. Per vaginam, the cervix was soft; otherwise nothing abnormal was felt. On withdrawing the finger, a little coloured discharge was upon it. Ruptured ectopic gestation was diagnosed, and the patient was at once admitted to hospital.

Operation.—Before operation, a uterine sound was passed, and showed the cavity from ½ to ¾ inch enlarged. Subumbilical median incision. On opening the peritoneal cavity, blood escaped freely. The intestines were distended, and there was commencing peritonitis. The Trendelenburg position was adopted, the hand was passed into the pelvis, and the left tube was found distended and brought into the incision. It was filled with blood clot, which protruded from the abdominal ostium, greatly distended (tubal abortion). The tube was exsected. As her condition admitted of it, the pelvis and loins were next cleaned of blood. Normal convalescence. Fig. 1 shows the distended tube filled with blood clot, with the placental villi protruding from its abdominal ostium. Below is the tubal mole, which was surrounding its ampullary end.

Case II.—A. W., æt. 31, seen in consultation with Dr. Gaston's *locum tenens.*

Previous History.—Always regular, had suffered at times from indigestion and sickness; one child thirteen years previously. After her confinement she suffered from weakness of the womb, for which she wore instruments for three years. No subsequent pregnancy.

Present Illness.—On June 17, 1905, she missed her period, and suffered subsequently from sickness, but, owing to previous indigestion, she did not suspect pregnancy. On July 7, attacked with very sudden and severe pain in the left iliac fossa, extending thence all over the abdomen. Next morning she was sick. I saw her in consultation at 2 p.m.

Condition at Consultation.—She was difficult to rouse, and complained in a very feeble voice of extreme thirst. She was absolutely blanched, covered with a cold, clammy sweat, and appeared moribund. No changes in the breasts. Abdomen not distended, but very tender all over. Dulness present in the flanks. Per vaginam, nothing abnormal felt, but pressure upwards, by the sides of the uterus, elicited great tenderness. Blood was present on the finger on withdrawal. Removed to hospital on a stretcher, where, under the influence of external warmth, saline injections and strychnine subcutaneously, she rallied, and laparotomy was performed through the left rectus about two hours after my first seeing her. On opening the abdomen, about 2 pints of fluid and clotted blood escaped. The left tube was grasped, some adhesions present separated, the tube and ovary brought into the wound and removed. The blood in the pelvis was quickly sponged out, and the abdomen closed. Operation lasted 20 minutes. Normal recovery. Fig. 2 shows the ampullary half of the tube distended with blood clot, and the small rupture about the middle of it stuffed with placental villi (*a*).

Case III.—C. I., æt. 20, sent into the Royal Portsmouth Hospital under my care by Dr. Palser, of Southsea, on September 21, 1905.

Previous History.—Always good health and regular, never previously pregnant, married April 22, 1905.

Present Illness.—Saw her last period at the end of July, had suffered from morning sickness, and tenderness in the breasts, and considered herself pregnant. Had latterly felt discomfort in her right iliac region on exertion. On August 20, she was suddenly doubled up with severe pain in the lower abdomen, felt faint, and was very sick. Noticed no vaginal discharge, and felt pretty well the next day. A fortnight later had a similar attack, and noticed a slight coloured discharge from the vagina, which lasted two days. She remained well till September 21, when she had a third similar attack and was admitted to hospital.

Condition on Admission.—She was a healthy young woman, with very fresh complexion, which somewhat obscured her anæmia, more especially as she was seen by artificial light. Though, however, she retained the fresh colour in her cheeks, her gums, lips, and conjunctivæ were bloodless. Tongue, dry; pulse, 132; temperature, 98°; abdomen slightly distended and very tender, especially in the right iliac fossa. Liver dulness present. Per vaginam, a boggy, ill-defined, and very tender swelling present in Douglas's pouch. No coloured discharge present on the finger on withdrawal. The diagnosis of ruptured ectopic gestation was made, and the patient was at once submitted to operation.

Operation.—Abdomen opened along outer border of right rectus. A large quantity of fluid blood escaped on incising the peritoneum. Right tube surrounded by a mass of blood clot brought up into the incision. The fœtus was observed alive floating in the amnotic cavity. The tube and fœtus were removed, clots were sponged out of the pelvis, and the abdomen closed. Recovery uneventful. Fig. 3 shows the tube enormously distended with blood clot, and stuffed with placental villi (*a*), while below (*b*) is seen the amnon, which, at the time of delivery, was filled with liquor amnii, and surrounded the fœtus.

Case IV.—A. C., æt. 35, seen in consultation with Dr. Marwood, of Southsea.

Previous History.—Always irregular, sometimes missing 5 or 6 weeks. No history of pelvic inflammation or vaginal discharge. Has two children, the youngest 3¾ years. Has not been pregnant since.

Present Illness.—On September 4, 1905, after a six weeks' interval, a discharge like the period appeared. No morning sickness. The discharge continued a fortnight, when she was seized suddenly with severe pain in the right groin, accompanied by faintness and vomiting. On September 22, she had a second similar attack, which continued off and on for a fortnight. She then had a week's respite, after which, October 13, she had a third attack, characterised by pain all over the abdomen, but especially severe in the right groin. She was then better again for a few days, but, during the first week in November, she began to suffer from bearing-down pains in the back.

PLATE X.

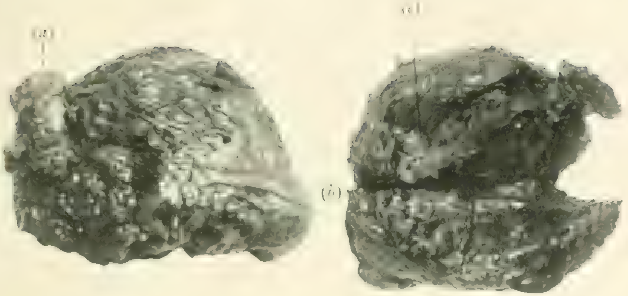

Fig. 1.

(a) Abdominal ostium of tube stuffed with blood clot, from which are seen protruding placental villi.

(b) Tubal mole which was attached to the abdominal ostium. In the centre is seen the remains of the fœtus (c).

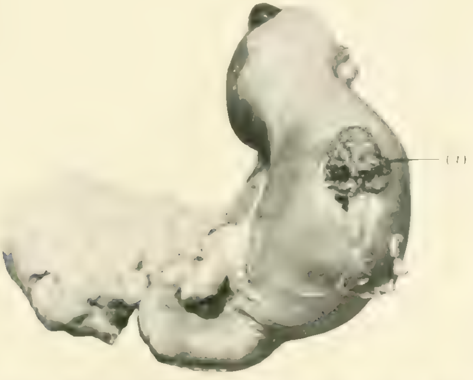

Fig. 2 *shows the ampullary half of the tube distended with blood clot, and the small rupture, about the middle of it, stuffed with placental villi (a).*

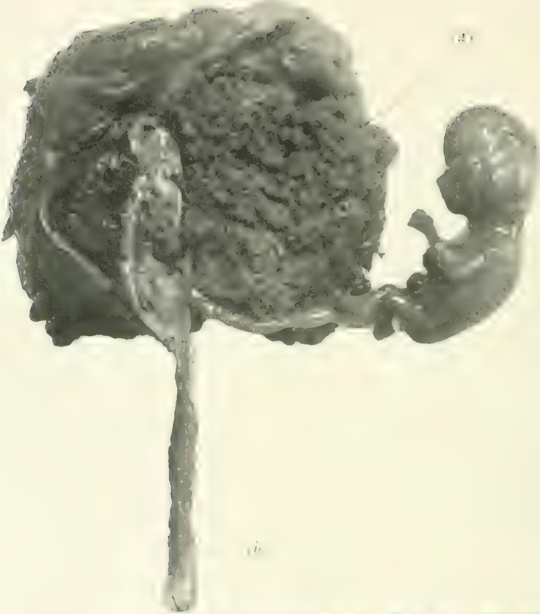

Fig. 3 *shows the tube enormously distended with blood clot, and stuffed with placental villi (a), while below (b) is seen the amnion, which, at the time of delivery, was filled with liquor amnii, and surrounded the fœtus.*

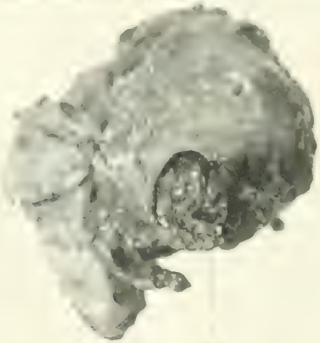

Fig. 4 *shows the ampullary half of the Fallopian tube distended with clot, which is protruding through the abdominal ostium, stuffed with placental villi (a). (Tubal abortion.)*

sickness, faintness, and restlessness. These symptoms recurred at intervals of 24 hours for 10 days, but, for a week previous to my seeing her, she had been freer of pain. Ever since September 4, the date of first noticing the vaginal discharge, she had had metrorrhagia, worse at the time of the attacks of pain.

Condition at Consultation, November 18.—Not anæmic. Morning sickness and changes in the breasts absent. Per vaginam, the uterus was low down and pushed to the left side: Os patulous: occupying Douglas's pouch and the right fornix was a tumour the size of a large orange. It was very tender, felt fluid, and appeared to be continuous with the uterus. No uterine souffle present. The sound passed 1 inch beyond the normal; the diagnosis was made of ectopic gestation with repeated hæmorrhages, and the patient was submitted to abdominal section on November 20.

Operation.—A tumour was found in Douglas's pouch, and spread over it, the Fallopian tube distended with blood clot. On incising the tumour, it contained greenish blood clot. It was enucleated, and the tube and ovary were exsected. Fig. 4 shows the ampullary half of the Fallopian tube distended with clot, which is protuding through the abdominal ostium, stuffed with placental villi (*a*) (tubal abortion). A gauze drain was inserted in Douglas's pouch, as there was a doubt as to the asepticity of the clot. Removed in 24 hours. Recovery uneventful, the highest temperature recorded after operation being 100°.

Case V. H. M., æt. 30, seen in consultation with Dr. Carling, of Southsea.

Previous History.—Two previous pregnancies, the last 9 years ago.

Present Illness.—On February 28, 1905, the period was a week overdue. On March 11, seized with severe pain in the lower abdomen: the bowels had not been opened for two days, and she was given an aperient. During the night, a little dark-coloured blood came from the vagina. Uterus was about the size of a two months' pregnancy, and the cervix was soft, and the os patulous. It was thought abortion was threatening. On March 13, retching and vomiting occurred, but there was no abdominal pain or tenderness. An enema of soap and water was given, the bowels acted well, and the sickness stopped. On the evening of the same day, she was seized suddenly with excruciating pain in the abdomen, and became deadly pale. At 11 p.m., I saw her in consultation with Dr. Carling.

Condition at Consultation.—Patient was blanched and very restless, pupils dilated, and pulse almost imperceptible; abdomen was very tender: per vaginam, nothing could be felt, but there was great tenderness on pressing upwards by the side of the uterus: a coloured discharge was on the finger on withdrawal: ruptured ectopic gestation was diagnosed. Although the patient was obviously in a very critical condition, it was the middle of the night, and there were no facilities for performing abdominal section in the house; she was, therefore, removed to hospital on a stretcher; the journey there tried her considerably, and when placed upon the operating table she was pulseless, and appeared at the point of death. Intravenous injection of saline solution revived her a little. Under eucaine, combined with a very little general anæsthetic, the abdomen was quickly opened. It was full of blood: the right tube ruptured was quickly seized and removed, and the abdomen at once closed, leaving the blood behind. The operation occupied but a few minutes, and the patient was no nearer death after it. Half an hour afterwards when I saw her in the ward, she had rallied considerably. The improvement, however, did not last, and she died at 8 a.m. on the following morning. The specimen was unfortunately lost, so that I am unable to complete the series of photographs, but it showed a rupture of the right tube into the general peritoneal cavity.

REMARKS.

On analysing the histories of these cases, we observe that the multiparæ were aged 31, 36, 35, and 30 respectively, the average age being 32¾ years; the primipara was aged 20. According to Bland-Sutton, the average age in 100 cases was as follows :—

Between 20 and 25 years	-	-	10 cases.
„ 25 „ 40 „	-	-	86 „
„ 40 „ 45 „	-	-	4 „

Presupposing a period of sterility, which is usually present, and reckoning that the period between 40 and 45 is the last flicker of the child-bearing period, we conclude that ectopic gestation would necessarily happen, in far the majority of cases, between the ages of 25 and 40. Of the multiparæ, none had been mothers of large families. Two of them had had but one previous pregnancy, the other two had been pregnant twice. Three out of the four gave a history of some previous pathological condition of the pelvic organs. Case I. always had a scanty period, and suffered from pains in the pelvis on exertion. Case II. had suffered after her confinement from "weakness of the womb," for which she had worn instruments for three years. At the operation pelvic adhesions were found. Case IV. had always been irregular. All of them furnished additional evidence of some abnormal condition by exhibiting a well-marked period of sterility. The first had not been pregnant for 11 years, the second for 13, the fourth for 3¾, and the fifth for 9 years. The histories of these cases, therefore, lend support to the theory that ectopic gestation usually owes its origin to some diseased condition of the tube interfering with the downward passage of the fertilised ovum into the uterus. The primipara, on the other hand, was a perfectly healthy young woman, and had always been regular and normal in her menstrual function. Her ectopic gestation probably furnishes an example of another recognised cause of this condition, viz., arrest of the ovum in a fœtal type of tube, or in a diverticulum from it.

Rupture is rarely deferred beyond the 10th to 12th week. All of these cases conformed to this rule as the following table shows :—

——	Missed Period.	First Abnormal Symptoms.	Rupture.
Case I. - -	March 19	April 2	May 3
Case II. - -	June 17	July 7	July 8
Case III - -	July (end)	August 20	September 21
Case IV. - -	August 21	September 4	September 17
Case V. - -	February 21	March 11	March 13

It will be seen that in every case, within three weeks of the missed period, the first symptom manifested itself, and rupture in the latest cases was not delayed beyond a month from the appearance of the first symptom.

As it is of the utmost importance that the condition should be recognised before rupture, or abortion into the peritoneal cavity, the symptoms should be clearly marked off into two distinct periods, and kept quite separate in the practitioner's mind, viz., those manifesting themselves ; *(a)* previous to rupture or abortion ; *(b)* when rupture or abortion occurs.

(*a*) *Symptoms previous to Rupture or Abortion.*—To begin with, patients may frequently, for various reasons, not suspect themselves pregnant. They have generally been sterile for a considerable period previously, so that their attention is diverted from pregnancy, and they are not on the look out for it. There may, in addition, be other misleading factors. For instance, though Case II., always previously regular, missed a period, she had been accustomed to attacks of indigestion and sickness, and did not therefore suspect pregnancy. Case IV. had always been irregular, sometimes missing five or six weeks. She had no morning sickness, or discomfort in her breasts, and did not, therefore, attach significance to the period being overdue. Case V. thought she had caught cold, and, after a sterile period of nine years, the idea of pregnancy never occurred to her. Cases I. and II., on the other hand, considered themselves pregnant. Though Case I. had been sterile for 11 years, she told me before operation that, from her own feelings, she was confident that she was pregnant. All of them had missed the period which should have appeared in the normal course of events. Three of them suffered in addition from sickness, the other two did not. None of the multiparæ had any feelings of discomfort or swelling in the breasts ; the primipara had. The constant primary symptom in all these cases, therefore, was the missed period ; the others were variable, and, for one reason or another, in three out of the five cases, did not direct the patients' thought to pregnancy. The next sign, in two of the patients, was a dark-coloured vaginal discharge, which continued up to the time of the first attack of pain. Case III. had felt pain in her right side on exertion. In the other two cases, the sign next after the missed period was sudden and acute pelvic pain. All experienced more than one attack of acute abdominal pain previous to the final catastrophe. Thus Case I. had four such attacks ; Case II. one attack the day before ; Case III. two attacks ; Case IV., which was a more chronic case than the others, and not quite in the same category, no less than eight attacks ; and Case V., two attacks.

It will be seen, therefore, that we have a very clear clinical picture, previous to the rupture or abortion, of a tubal gestation. The patient misses a period, and probably has, in addition, some, if not all, of the concomitant signs of early pregnancy. Enquiry will very likely elicit the fact of a previous period of sterility. She next either sees a dark-coloured vaginal discharge, or feels crampy pains in the lower abdomen. If the discharge comes first, it is followed, at a short interval, by pain. If she feels the pain first, it is accompanied, or soon followed, by the discharge. It is evident that the foregoing symptoms are not unlike those of impending uterine abortion, and, if a patient presents herself to the doctor with such a history, the familiar accident, abortion, is likely to come uppermost in his mind, and not the exceptional accident, ectopic gestation, of which he may have never had any previous experience. It should, therefore, become a canon of midwifery practice "*that, in every case in which symptoms* *suggestive of abortion present themselves to the practitioner, ectopic gestation should be excluded or confirmed.*" For this purpose, an examination should be at once directed to two points. An enlarged tube should be looked for, and the vaginal discharge should be submitted to expert microscopical examination. If an enlarged tube is discovered, and decidual cells, but not chorionic villi, are found in the discharge, the diagnosis is practically certain.

If, on the other hand, chorionic villi are discovered in the discharge, it will be evidence that the pregnancy is uterine, in which case the enlarged tube would probably be due to some other condition. But the possibility of the simultaneous occurrence of intra-uterine and extra-uterine pregnancy, of which numerous cases have been recorded, must be borne in mind. The intention of the present paper is not, however, to multiply difficulties by drawing attention to rare and exceptional conditions, which may lead the most experienced astray, but to emphasise the importance of bearing in mind that every case, presenting what appear to be symptoms of impending uterine abortion, may in reality be the subject of ectopic gestation, and of insisting on the necessity of excluding the latter before assuming the former. To recognise the condition at this stage, before rupture occurs, is the essential factor. The percentage of cures would then be, with aseptic care, practically one hundred. On the other hand, the usual concluding stage of this condition, the rupture, or abortion into the peritoneal cavity, must always be fraught with danger to life, and the most prompt and skilful diagnosis and treatment may fail to avert a catastrophe.

(*b*) *Symptoms when Rupture or Abortion occurs.*—It is equally important that the clinical picture of this stage should be familiar to every practitioner, for, though the ideal treatment is the removal of the tube before rupture, even after this accident has happened, prompt diagnosis and treatment give remarkably favourable results. The symptoms of onset are those common to acute abdominal attacks generally, viz., sudden and severe pain in the abdomen accompanied with vomiting and collapse. The general symptoms are those of internal hæmorrhage. The great thirst, faintness, and restlessness, the oppression at the heart, the "air hunger," the blanched face, the bloodless lips and conjunctivæ, the almost imperceptible pulse, and dry tongue, point the way. If the patient is seen in this condition, and the diagnosis of internal hæmorrhage, in contradistinction to perforation, is made, a little further enquiry will make the diagnosis clear. The history, previous to rupture, will put the practitioner on the track at once. An examination will reveal the abdomen possibly distended, always extremely tender. Dulness may be present in the flanks, as in the first two cases here recorded, or it may be entirely absent. Liver dulness is present. Per vaginam, very little may be felt. It must be remembered that these patients are usually extremely ill, and an exhaustive examination is often out of the question, indeed, it might do much harm by exciting fresh hæmorrhage. The cervix may be felt to be soft. Pressure upwards around the uterus will usually elicit great tenderness. On withdrawing the finger, a little dark blood will probably be upon it, but may be absent, as in Case III. Sometimes a boggy, ill-defined swelling will be felt in Douglas's pouch, which is very tender ; or, especially if the case is more chronic and inflammatory conditions have been added, the uterus may be pushed out of place by a swelling which appears to be continuous with it. If the uterine sound is now passed, it will be found enlarged half an inch to one inch. It will be observed that the clinical picture is clear. Though all the characteristic features may not be present in every case, the diagnosis is, I think, easier than that of most acute abdominal affections. In all of the cases related here, the diagnosis was confidently made beforehand, and turned out to be correct. To avoid errors arising from cocksureness, a few questions should be asked, and examination directed to exclude other conditions, with which it might be confounded. In abdominal diagnosis, however apparently clear the picture, the method of exclusion should always be practised.

With regard to treatment, there is little room for divergence of opinion. If diagnosed early and before rupture, the condition should be dealt with surgically by immediate removal of the tube and its contents. This is the ideal treatment, but it is unfortunately too seldom accomplished. When rupture has occurred, mmediate operation is almost universally the only line or safety. Although many cases probably recover without the condition being suspected, or the diagnosis made, the accident is one of the greatest gravity, and demands prompt interference.* There are only two exceptions, in which the propriety of immediate operation is open to question. The first is when the patient is in profound collapse. Case IV., the only fatal one in this series, was an example of this condition. She appeared to be, and indeed was, almost moribund when placed on the table, and it might have been wise to temporise, and hope for a more favourable opportunity to operate. But, although it is easy to formulate recipes for emergencies from the armchair, when the surgeon is face to face with a patient who appears to be slipping through his fingers from hæmorrhage, and knows, moreover, exactly where the bleeding point is, and that he can secure it in a few moments, it is difficult to stand easy and wait. At all events if he did so, and the patient died, he would inevitably regret his decision, or rather indecision. It seems that there are very sound reasons for not waiting. If the surgeon is confident in his own mind of the diagnosis, he knows exactly what he has to do, and he knows that it will take him very little time to do it. It is not as if he were going to spend half an hour in locating an unknown bleeding point in the abdomen. He knows where to go for the bleeding point, and that he can have it under control within five minutes of his taking the knife in his hand. It would seem, therefore, to be good practice to secure it under local anæsthesia, after rallying his patient by saline venous injection. Case V., though she eventually succumbed, was not the least bit the worse for the operation, which occupied a very few moments, and she had visibly rallied half an hour afterwards. My own inclination, therefore, is in favour of immediately securing the bleeding point rather than of trusting to the uncertainty of delay. It is impossible to dogmatise. Each case must be left to the decision of the operator, who is faced with the responsibility of it. Any surgeon, who has saved one of these patients from the very jaws of death by either course, will be able to be wise after the event, and to justify his action. Any surgeon, who has failed, will perhaps regret that he did not adopt the alternative; though I think that the certainty of being able to secure the bleeding point, by a very rapid procedure, is a strong argument in favour of adhering to an established surgical principle, viz., to secure a bleeding vessel as soon as possible.

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* A. Martin has shown that 36·9 per cent. out of 265 cases recovered under expectant treatment, but that 76·7 per cent. out of 575 cases recovered under operative treatment.

The second possible indication for delay is, under certain conditions, when a well-marked hæmatocele has formed. Case IV. was an example of this kind, and the following Case VI. is another, though they differ somewhat, this difference determining the treatment that was adopted in each instance.

Case VI.—S. S., æt. 32, sent to me at the Royal Portsmouth Hospital by Dr. Mayhew, of Southsea, on March 11, 1905.

Previous History.—Married 6 years, never previously pregnant. Before marriage not always regular, but since always so.

Present Illness.—In January 1905, had a scanty period. Soon afterwards, a coloured vaginal discharge appeared, which continued up to a few days before admission. Morning sickness was present. On February 11, she was attacked with severe pain in the lower abdomen, accompanied by retching. Four days afterwards, experienced a slight return, and, a week later, a similar attack to the first, which lasted all day. On March 25, felt sudden and severe pain in the right groin, and from that time suffered from pain in the lower abdomen, off and on up to shortly before admission. Retention of urine on two occasions. Constipation present, with great pain when the bowels acted. For four days previous to admission to hospital (March 11), the pain and discharge had left her.

Condition on Admission.—The breasts presented no visible changes, but a little secretion was squeezed from the right nipple. Abdominal examination negative. Per vaginam, a hard tumour (blood + inflammatory exudation) occupied Douglas's pouch. It was very tender, and flattened out the rectum, from which there was an escape of some blood and mucus. The cervix was pushed upwards and in front of the tumour. Temperature 99°.

As her distressing symptoms (pain and discharge) had left for four days previous to admission, she was kept in bed for observation. She went on obviously improving from day to day. No operation was consequently performed, and, in six weeks, the tumour was represented by a small hard lump, about the size of a walnut. In uncomplicated encapsulated hæmatocele, expectant treatment is advocated by Zweifel and others, though most modern gynæcologists advise operation. Individual cases may, with advantage, be decided on their merits. Where, as in this case, the patient when first seen is obviously getting better, and the lump diminishing from day to day, with a normal temperature, there seems to be no indication to interfere. On the other hand, in Case IV., an example also of encapsulated hæmatocele, the symptoms were active and clearly indicated intervention. Old cases, in which there is no fear of severe hæmorrhage, may be treated extra-peritoneally, either above Poupart's ligament after pushing up the peritoneum, or by the vaginal route. But the surgeon should be prepared to open the abdomen at once, in the event of severe or uncontrollable hæmorrhage.

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DEMONSTRATIONS ON DISEASES OF THE NERVOUS SYSTEM.[1]

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II.—DISSEMINATED SCLEROSIS.

A YOUNG woman, aged 22, first sought advice, a little over twelve months ago, on account of eye troubles. She complained of double vision, and stated that she had had two other similar attacks during the previous two years.

This diplopia proved to be due to weakness of the external rectus of the right side, and further examination showed that the optic nerve also on this side was distinctly paler than it should be. General power of vision was good, but there was a small central scotoma for green in the right field.

I was then asked to see her, and some other important points in her history were obtained.

So long as five years ago, she had an attack of numbness in the right arm, which prevented her from working for some weeks, but which eventually disappeared quite suddenly. Since then she has had attacks of a similar nature in the lower limbs, so that one leg has sometimes dragged.

Any doubt as to the serious nature of the disease underlying the symptoms in this case will have already been dispelled by a knowledge of the existence of the pallor of the right optic nerve and weakness of the right external rectus, but, had these conditions not been present, there are others, which conclusively show that the varying symptoms, however superficially suggestive of functional disorder, will not, when investigated, bear that interpretation. For, at the time the patient first came, it was possible to show further that the orbicularis palpebrarum and the corrugator supercilii of the right side were weak; that the knee-jerks were increased, and that the plantar reflexes gave the extensor response, while the superficial abdominal reflexes were absent. It also transpired that there was occasional difficulty in retaining the water.

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[1] From the Out-patient Department of the Middlesex Hospital.

Now this patient affords an excellent lesson as regards the way in which the signs vary in the early stages of disseminated sclerosis.

There is the record as to how the numbness and weakness of the limbs came and went, and opportunities have been afforded of seeing how the symptoms have varied since she has been attending the hospital. Sometimes she seems almost as though she had nothing the matter with her; at others she is weak, and perhaps also has nystagmus and diplopia. A month ago she was feeling weak generally, now she feels stronger, but is again bothered with the double vision.

It is this variability of the symptoms which makes disseminated sclerosis so liable to be confused with functional disorders when it first begins. In this instance there was no difficulty, for from the first there were numerous signs, which certainly depended upon organic disease, but had the patient been seen earlier still, when she only complained of temporary numbness of one arm, which disappeared as suddenly and mysteriously as it came, it is quite possible that the most careful examination would have failed to reveal anything of a positive nature, and the temptation to write it down functional would certainly have been great Where the history is suspicious, and the signs not trustworthy, it is wise, if possible, not to express a decided opinion until a chance of observing the patient for a time has been afforded. The signs, one or more of which are most likely to be of service, are weakness of an ocular muscle, double vision, slight nystagmus, pallor of one or both optic nerves, extensor response of the plantar reflex, and perhaps some complaint of a difficulty in controlling the sphincters.

On looking at this patient, it is at once observed how different the signs are from those in which the cases are of the classical variety, characterised chiefly by spastic gait, ankle clonus, intentional tremor, nystagmus and staccato speech.

While the alterations which finally take place in the nervous system are immutable, it is obvious that the earlier ones are not so, and it seems likely that exudation of variable degree precedes the formation of connective tissue patches.

What with the variability of the symptoms at the beginning, and the lack of any certain clinical picture later, owing to the different distribution of the patches in different cases, it will be easy to understand how errors in diagnosis are likely to arise.

If the disease is considered, so far as possible, at its different stages, the difficulties that are likely to occur can be summarised somewhat as follows :—

1. During the early part of the disorder, the symptoms may vary so much that there is danger of diagnosing *Hysteria*, or some other functional disease.

2. As the patches of sclerosis develop and become more permanent, they are very likely to occur in the course of the crossed pyramidal tracts. Hence a patient may come complaining of a weakness of the legs, which appears to depend upon a degenerative process of the lateral columns.

In the absence of other signs, the diagnosis of spastic paraplegia will be rightly made, but the important question arises as to whether it is to be regarded as *Primary Spastic Paraplegia*, the prognosis of which is certainly better than that of disseminated sclerosis. While time may be required before deciding, there should be considerable hesitation in diagnosing primary spastic paraplegia, for experience shows that the greater number of patients, who for a time are apparently suffering from this disorder, turn out in the end to be victims of disseminated sclerosis.

3. The addition of some patches in the posterior columns may cause ataxia, and so give rise to the diagnosis of *Ataxic Paraplegia*, and the remarks concerning the fate of cases of seemingly primary lateral sclerosis, apply equally to ataxic paraplegia.

4. When the patches are more freely disseminated, but still do not give rise to the more typical symptoms, the disease may be confused with *General Paralysis of the Insane*, *Friedreich's Disease*, *Cerebellar Disease*, and, in children, with *Cerebral Diplegia*.

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ON THE RELATIONSHIP BETWEEN SIMPLE POSTERIOR BASIC MENINGITIS AND CEREBRO-SPINAL FEVER.*

By FREDK. LANGMEAD, M.D., M.R.C.P.,

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SINCE attention was first called to the disease, which we now know by the name of posterior basic meningitis, by Dr. Gee and Sir Thomas Barlow[1] in 1878, our knowledge of this condition has gradually accumulated. The constancy of the symptoms and post-mortem appearances was emphasised by Dr. Carr[2] in 1897, and his paper and that of Sir Thomas Barlow and Dr. Lees in Clifford Allbutt's *System of Medicine* have done much to establish it as a clinical entity. A turning point was reached in 1898, when Dr. Still[3] demonstrated as the cause, the diplococcus, which has since been associated with his name. The close resemblance, which this organism bears to the diplococcus of Weichselbaum, at once raised the question: Was posterior basic meningitis a disease *sui generis*, or was it only a variety of so-called spotted fever, a condition already rich in variations? Many have already accepted the latter alternative as the true solution, but that the question is still an open one may be gathered by reference to our modern text-books. In these, the two conditions are either treated as separate diseases, or, if a description of posterior basic meningitis finds a place under the heading "cerebro-spinal fever," it is there only on approbation, as it were, and is not included by the author with any degree of assurance. Also at least one writer (Hildesheim[4]), who speaks with no uncertain voice, would keep these two conditions definitely distinct. I am with the flowing tide in believing that posterior basic meningitis is sporadic cerebro-spinal fever, modified, it is true, but not differing from it more that one epidemic differs from another.

The object of this paper is to put before you the foundations for that conviction, and, in order to do so, I shall first give a short *résumé* of the more salient points of the clinical and pathological features of posterior basic meningitis as it is met with in infants. With these I shall compare the symptoms of the disease as it occurs in older children, and shall try to show that posterior basic meningitis, in children older than usual, forms a link between the disease, as it is found in infants, and cerebro-spinal fever.

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* Including part of a paper read before the St. Mary's Hospital Medical Society.

Posterior basic meningitis, as usually described, is a disease of early infancy, and but seldom occurs in children over two.

Sir Thomas Barlow and Dr. Lees[5] state that, in a series of 110 cases, 84 were in children under 12 months old. Its onset is usually sudden. An infant, previously in good health, or showing at the most evidences of catarrh of the nose or air passages, is seized with vomiting, and is feverish, it seems "queer," as the mother puts it, it screams without apparent cause, and possibly has a convulsion. In a few hours or days, the more characteristic features of the disease become manifest. The most striking and important of these is retraction of the head. It is no mere falling back, due to weakness; but an active contraction of the nuchal muscles, for, if these are felt, they will be found to stand out like rigid bands. In most cases, the retraction is limited to the head, but, in some, an associated contraction of the back muscles produces a true opisthotonos, so that the shoulders are also bent back, producing a most remarkable deformity. The chest bulges anteriorly, whilst in 2 cases which I have seen, it was necessary to place a pad between the occiput and sacrum, to prevent ulceration. The arms are fixed in a condition of rigid extension, and frequently superpronation, so that the palms look outward. The fists are clenched. The legs, too, are rigidly outstretched, and often crossed; the feet arched, with the toes pointing downwards. The spasm, which produces this deformity, may be continuous, but is more often imperfectly sustained, the remission periods of flaccidity varying widely in duration and completeness. Perhaps next in importance is the state of the eyes. About 33 per cent. of these infants are quite blind, a blindness which is central and not peripheral, since careful examination of the disc reveals optic neuritis only in a very few. The pupils are usually dilated, and the upper lids retracted, allowing a margin of sclerotic to be seen, a combination which gives a curious blank, staring look to the patient. Squints are not common, except in the later stages of fatal cases. Sucking movements of the lips are sometimes seen, and frequently a grinding and munching of the teeth, which closely simulates the movements of a horse champing at its bit. The mental condition is one of subconsciousness rather than unconsciousness, and attempted flexion of the head is usually followed by a loud querulous cry, with evident signs of pain. In cases going to the bad, this is replaced by a deepening coma.

Convulsions, clonic or tonic, may occur, but are not so common as in other forms of meningitis. Vomiting, although by no means characteristic, is a very troublesome and constant feature. It begins early, is explosive in character, and is often frequently repeated. Coupled with the difficulty in feeding, which the rigidity and spasms cause, it leads to a degree of emaciation in protracted cases, hardly equalled even in a children's hospital. The mental picture of an infant, lying with arched back, retracted head, with well dilated, staring but sightless eyes, stiffened by muscular spasm, so that it can be raised *en bloc* by one hand under the occiput, and another under the heels, emaciated, so that it looks as though it were made up of a bony skeleton, wrapped in a shroud of skin, is one not readily forgotten. The temperature may be raised at the onset to 102° or 103° F., but, later, is usually only slightly above the normal, and has nothing distinctive about it. In acute severe cases, it may, however, be high and irregular, and hyperpyrexia is common just before death. The breathing is irregular; sometimes it is typically Cheyne-Stokes in character, but more often the respirations are grouped in batches of threes, fours, or fives. In a few cases a dusky swelling occurs about the joints.

Complications.—The most frequent and fatal is hydrocephalus. In nearly every case, which withstands the first onslaught of the disease, a bulging, tense, fontanelle can be recognised, and even children, who have apparently recovered, generally show an increasing growth of the head, or evidence of concealed hydrocephalus, by convulsions, insanity, paralysis, incontinence, or low moral tone (Hildesheim[6]).

Pathology.—The name, by which the disease is known, gives the clue to the most constant lesion which it produces.

At the posterior part of the base of the brain, gluing the medulla to the under surface of the cerebellum, and filling up the cisterna magna, is a diamond-shaped area of yellow fibrino-purulent exudate, beneath the arachnoid membrane. Although inflammatory changes may be confined to this region in the great bulk of cases, they are not so, but spread forward on the base as far as the interpeduncular space, and the tips of the temporal lobes, inward to the ventricles, and downward to the spinal membranes, producing similar patches of exudate, generally on the posterior surfaces of the cervical and lumbar enlargements. It has been said that involvement of the vertex is very rare ; this is certainly an exaggeration, for among the last 50 cases at Great Ormond Street, no less than 19 showed some vertical meningitis. That it never occurs purely, or primarily, on the vertex, is probably nearer the truth.

Hydrocephalus of greater or less extent is almost always present.

Bacteriology.—Dr. Still,[7] in 1898, obtained from the cerebro-spinal fluid, and grew in pure culture, a small diplococcus in several successive cases. Time has testified as to the value of this work, and the causal relation of this organism to posterior basic meningitis is now not doubted. With due care it can be grown from the cerebro-spinal fluid obtained by lumbar puncture, or after death, in practically every case, during the active stage. It may be known by the following attributes :—It is a small organism, measuring only $1 \cdot 5\ \mu$ in length and occurs in pairs. It resembles the gonococcus, in that the adjacent sides of each coccus are flattened, and two diplococci are frequently seen together, giving a tebracoccus form. It may be found free, or within the cells of the exudate. It is aerobic, and grows readily on agar-agar, glycerine agar, or blood agar, and in broth or milk. On blood serum, the growth is scanty. It does not stain by Gram's method. It was said to very closely resemble the diplococcus of Weichselbaum, the cause of epidemic cerebro-spinal meningitis, but to differ from it in certain cultural properties. I will give these differences in detail, because, by this fine thread, hangs most of the bacteriological evidence, which keeps posterior basic meningitis from being enrolled among the variations of cerebro-spinal fever, evidence, which I hope to show later, is quite inadequate, and has been falsified by more recent work.

Briefly, the differences were said to be *readier growth* and *greater vitality*.

The diplococcus of Still grows readily on agar, and, in four hours, the culture can be recognised as small round, slightly raised colonies, looking like drops of gum. The diplococcus of Weichselbaum was said to grow with extreme difficulty, and then in the form of a very fine dust. The diplococcus of Still grows readily in broth, that of Weichselbaum was said not to grow in it at all. The diplococcus of Still could be kept alive for 24–34 days on agar, and for 53 days on blood agar. The diplococcus of Weichselbaum was said to quickly die. These were the features on which the separate identity of Dr. Still's organism rested, features which he himself thought were "the result of natural variation, and represented rather a modification of characteristics than a distinction in kind."

From what has been said, it is at once evident that there is a certain similarity between the two diseases under consideration. In both there is an initial coryza, leading to irritability and headache ; in both there are rigidity, head retraction, strabismus, spasms (tonic or clonic), and in both hydrocephalus is the most common sequela.

Let us see what are the chief differences. They arrange themselves naturally into three groups, clinical, pathological, and bacteriological. The synonym for epidemic cerebro-spinal fever—spotted fever—at once suggests a clinical distinction, the presence or absence of a petechial rash. But spotted fever is a bad name. Many epidemics have been described in which no rash was present, and even when present the proportion of cases, in which it is found, is less than is generally supposed. In the recent epidemic in New York,[8] it occurred in one-fifth. Herpes, which in frequency is next to purpura, was present in one-sixth of the cases.

In the posterior basic meningitis of infants, rashes, it is true, are very rare, but they do occasionally appear. Other distinctions are found in the eyes ; amaurosis is not a feature of cerebro-spinal fever, as it is of posterior basic meningitis. On the other hand, optic neuritis is much commoner in the epidemic form (6 in 40 according to Randolph[9]). Lastly, posterior basic meningitis was said not to occur epidemically.

On the assumption that perhaps age has something to do with the differences in clinical symptoms, I have put together the record of posterior basic meningitis in children, over 3½ years, who have been admitted into the Hospital for Sick Children, since the bacteriology has been worked out. They form but a small group, 10 in all, but afford a very interesting comparison. So far as they go, they strongly support the identity of the two diseases. They bear much greater resemblance to sporadic cases of cerebro-spinal fever than to posterior basic meningitis in infants. Only 1 of the 10 was blind, whilst 5, *i.e.*, half, had optic neuritis, 2 of the 10 had herpes. Another feature, which is very uncommon in posterior basic meningitis in infants, was present in 8 of these 10 cases. I refer to a swinging irregular temperature, rapidly rising and falling through a wide range, so that the curve is like that of a pyæmia. With the rises of temperature, there is frequently increased restlessness and rigidity, possibly rigors, headache, and greater mental disturbance, and sometimes also vomiting. These accessions of fever were described, in November 1904, by Box, in a paper read before the Clinical Society, having the title "The Crises of Posterior Basic Meningitis." It is noteworthy that the youngest of his series was 9 years old. Now *this* is a feature, which has given its name to one of the classical forms of epidemic cerebro-spinal fever, viz., the intermittent form, described by Von Ziemssen and Stillé.[10] These points are shown in the following table:—

COMPARISON OF CLINICAL FEATURES.

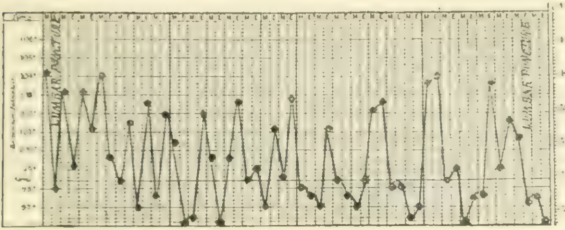
—	Rash.	Temperature.	Optic Neuritis.	Amaurosis.
Posterior basic meningitis in infants.	Very rarely	Usually slight; higher and irregular if fatal.	3 in 42 = 7 per cent. (Barlow & Lees.)	At least ⅓.
Posterior basic meningitis in older children.	2 in 10 herpes.	8 in 10 irregularly intermittent.	5 in 10 = 50 per cent.	1 in 10.
Cerebro-spinal fever.	6 in 30 purpuric. 5 in 30 herpes. New York epidemic.	No fixed type: but one form "intermittent."	6 in 40 = 15 per cent. (Randolph.)	Very rarely.

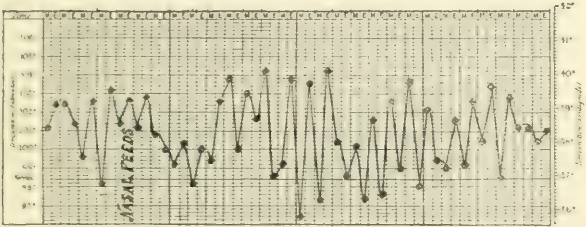
I will not trouble you with the notes of all these cases, but will make the last two serve as examples of the series.

Case 1.—A girl, aged 4 years and 10 months. The history was that, on April 11, 1904, she woke up in the morning and asked for water, which she vomited immediately, and then became delirious. Before this she had been perfectly well. She then complained of headache, and cried out continually. She vomited everything that she swallowed, and as this and the headache persisted, she was taken to the hospital. There was nothing of importance, either in the previous or family history. When examined after admission, she was seen to be lying with her head greatly retracted, and spine curved, producing opisthotonos. The eyes were closed, the teeth clenched, and the lips drawn apart. She kept up a continual moaning, and, at times, was very restless. Vomiting was occasional. The pupils were dilated. Reflexes were all lively. The plantar response was extensive. Kernig's sign was present. There was neither blindness nor squint. She was dazed, but not unconscious. Examination of the heart and lungs revealed no abnormality. No urinary change was present. She remained under observation, in very much the same condition, for seven weeks, at one time being rigid, with great head retraction and opisthotonos, during which she was partly conscious, at another being bright, and quite aware of her surroundings, and free from muscular spasm. An important feature was the temperature, which was intermittent in type, and for several successive days remarkably regular in its accessions, attaining its height regularly at 3 a.m., and its lowest point at about 11 a.m. At the height of the fever the restlessness, moaning, stupor, and spasm increased. Its daily variation was frequently from 97° to 104° F. The vomiting stopped on the fourth day after admission. She was very difficult to feed, and nasal feeding had to be resorted to, and continued without cessation every four hours for over seven weeks.

In spite of this, she became extremely emaciated, and progressively more feeble, so that her limbs were tremulous when lifted, and her speech was scarcely audible, although quite coherent. Slight optic neuritis developed. As the condition was practically unchanged, iodide of potassium was tried as a last resource, with excellent results. Three grains were given four times a day. Four days later the temperature had dropped to normal, and she was brighter and sitting up, nursing her doll. From this time onwards recovery was rapid, the optic neuritis disappeared, and she gained flesh quickly. Lumbar puncture was successful, but no organism was grown. There was an excess of polymorphonuclear leucocytes. When seen, six months later, she was apparently a normal child.

Case 2 was that of a boy, aged 4 years and 3 months. On November 10, 1904, he was sent home from school because he was complaining of headache, and his head was noticed to be drawn back. He had been sitting, playing with some beads, and had suddenly cried out. He vomited after getting home, and, from that day, until admission, on November 24, he had frequent severe headaches with vomiting and thirst. On admission, he was found to be lying with head retracted, and any attempt to move increased the spasm. On trying to bend the head forward by a hand under the occiput, the whole body was lifted, and he cried out with pain. He was quite conscious and not blind. The pupillary reflex was normal. There was no squint. The knee jerks were active. The plantar response was flexor. The discs were normal. There was no rash. From admission until December 20, about one month later, the temperature pursued a very up and down course, varying each day from 96° to 97° F. to 103° or 104° F., with occasional added irregularities. The rises were sharp, frequently associated with a rigor, and always with great restlessness, screaming, headache, and thirst. After about an hour, the temperature would suddenly drop, and the child again become rational, remaining quiet until the next rise. These accessions were regular in their onset, coming on two hours later each day. His condition remained much the same as that in which he was admitted, except that he became progressively thinner, and somewhat more drowsy. He had neither the characteristic stare nor blindness of a posterior basic meningitis, and was never unconscious. The fundi remained normal. Towards the end of this period, he became very feeble and emaciated, and the slightest movement was accompanied by tremor. No change was recognised in heart, lungs, or abdomen. By lumbar puncture, a turbid fluid was withdrawn, containing an excess of polymorphonuclear leucocytes, but sterile. On December 21st, 5 grains of potassium iodide were given to him every 6 hours, and from that time onwards, the temperature remained normal, and convalescence was established. On the 21st, lumbar puncture proved the diagnosis, yielding a growth of the diplococcus intracellularis.





Too much emphasis must not be laid on so few as ten cases, but, looking at them with this reserve, they do constitute a bridge, between posterior basic meningitis in infants, and cerebro-spinal fever, since, although they were diagnosed as posterior basic, and yielded an organism in most cases indistinguishable from that of Dr. Still, they really more closely resembled the latter.

To the objection that posterior basic meningitis does not occur in epidemics, an answer is found in a recent paper by Koplik.[11] He records a series of cases, showing all the characters of this disease, which occurred during the recent epidemic of cerebro-spinal fever in New York. Perhaps, too, our own cases are not so isolated as we imagine. To follow out this suggestion, it would be necessary to examine the records of all the London Hospitals, an impossible task, but a study of those at the Hospital for Sick Children lends support to it. Most of the cases came from the same district, but more important than this, many cases of the same year came from adjoining streets, whilst, in two or three instances, I found that one case, from one street, was quickly followed by another from the same.

The pathological and bacteriological differences, I have already to some extent indicated. The exudate, in posterior-basic meningitis, is said to be at the posterior base, and about the cord; that of cerebro-spinal fever, on vertex, base and cord alike; but, on the one hand, in 19 cases out of 50, there was exudate on the vertex, and in 34 out of 50 in the ventricles, in bodies of children, who had died of the former disease, and, on the other, in cerebro-spinal fever, the posterior base bears the brunt of the infection.

Bacteriologically the gap is but narrow, but, such as it is, it is being filled up by recent work. Netter[12] states that he has grown the diplococcus of Weichselbaum on broth, and that the same organism has been cultivated in an oven for more than four years, so that the two chief cultural differences no longer hold good. Moreover, Hunter and Nuthall,[13] obtained by lumbar puncture, from seven patients, suffering from posterior basic meningitis, organisms absolutely identical with that described by Weichselbaum, and from three others, organisms, which resembled the description of the diplococcus meningitidis, as given by Jaeger and Heubner. In conclusion, I will add the notes of a case, admitted in February 1905, which was clinically one of the most virulent forms of epidemic cerebro-spinal fever, but bacteriologically was in every way indistinguishable from posterior basic meningitis. It was that of an infant aged 11 months. She had been wasting, and had had a cough for two months. No more definite history of the onset of the meningitis could be obtained, than that drowsiness had been noticed for two or three days. When admitted she was extremely ill, and was partly conscious. She vomited frequently. The respirations were Cheyne-Stokes in character. No squint was present. The knee-jerks were obtained. There was no paralysis of cranial or spinal nerves. Two days later, she developed a few purpuric spots on the eyelids and cheeks. The rash rapidly spread, until there was a purpuric mottling all over the face, on the arms and legs, and slightly on the trunk. A few spots were seen on the buccal mucous membrane. She developed a squint, and, after two convulsions, became comatose and died three days after admission. Post-mortem examination revealed a general purulent meningitis of the cerebral and spinal meninges. The exudate, which elsewhere was creamy and fluid, in the posterior basic region and spinal meninges, was thicker and denser. In the posterior basic region, too, it was in greatest abundance. Pus was present in both ears. Films from the cerebral meninges, spinal meninges, and middle ear were examined by Dr. Graham Forbes, who found large numbers of non-Gram staining intracellular diplococci, and cultures from the blood, and from the pus in these situations, all grew organisms, in every way resembling the diplococcus of posterior basic meningitis. To sum up :—

1. Although there are certain clinical distinctions between posterior basic meningitis and cerebro-spinal fever, yet by an appeal to the record of the former in older children, those distinctions are lost.

2. Posterior basic meningitis can occur in epidemics.

3. The pathological lesions of this disease have been too narrowly defined.

4. The slender bacteriological distinctions are being nullified by recent experiment.

5. The diplococcus, as described by Dr. Still, has been isolated from the blood of a case of malignant epidemic cerebro-spinal fever.

If this view of the identity of posterior basic meningitis and cerebro-spinal fever is correct, it means that we have always with us, organisms, which under certain imperfectly understood conditions, probably of faulty hygiene, are capable of attaining a high degree of virulence, and producing the epidemic form.

My thanks are due to the Physicians at the Hospital for Sick Children, by whose kindness I have been allowed to use the records on which this paper is based.

[1] Gee and Barlow : *Reports of St. Barts. Hosp.*, 1878.
[2] Carr : *Trans. Medico-Chirurg. Soc.*, 1897.
[3] Still : *Trans. Path. Soc.*, 1898.
[4] Hildesheim : *Lancet*, 1905.
[5] Barlow and Lees : Clifford Allbutt's *System of Medicine.*
[6] Hildesheim : THE PRACTITIONER, August and September, 1905.
[7] Still : *loc. cit.*
[8] Foster : *Am. Journ. of Med. Sci.*, 1905 (January to June).
[9] Randolph, quoted by Osler : *Principles and Practice of Medicine.*
[10] V. Ziemssen and Stillé, quoted by Osler : *loc. cit.*
[11] Koplik : *Am. Journ. of Med. Sci.*, 1905 (January to June).
[12] Netter : *Supplement to Twentieth Century Practice of Medicine*, 1903.
[13] Hunter and Nuthall, quoted by Netter : *loc. cit.*

WHAT IS FEVER?

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If there is anything, upon which we, as clinicians, particularly pride ourselves, it is our knowledge of fever.

The thermometer has become as indispensable a part of our equipment as the prescription-blank, or the lancet of former generations. Our first procedure, become almost instinctive in a case showing any degree of acuteness, is to slip it under the tongue, and read off the position of its silver bar. Should that reading be above 100°, we look grave and say that the patient has fever, and usually proceed to reduce it, too often with one of the innumerable "Antis", which have been fully advertised these fifteen years past. We know perfectly well what fever is, what it indicates, and what to do for it, and yet, if anyone were to ask us what the meaning of this rise of temperature is; or what the mechanism of its production; we should find ourselves floundering at once. There is no better illustration of how painfully close even our most recent knowledge comes to being a mere definition of our ignorance.

Nor are these questions mere academic or transcendental ones; on the contrary, they are of the gravest and most practical importance. Upon our answer to them depends our whole attitude toward, and consequent treatment of, this departure from the normal. Ten years ago, we were making a fetich of the fever thermometer; while admitting that the signs of pulse, respiration, pain, etc., might or might not be significant of morbid states, here was a criterion that never failed us. Whenever the sacred level of 98·6° was seriously exceeded, there was disease at once, serious disease which was to be promptly combated by modern, or ancient, antiphlogistic methods. Fever was always and everywhere to be attacked, and reduced by every reasonable means at our disposal. Like Carthage "*delenda est.*" In other words we were treating a symptom as a cause. Fortunately, the reaction has set in, and we are beginning to question whether, in fighting indiscriminately against fever, we have not at times been attacking a friend instead of an enemy; whether the means used to reduce it have not done so by reducing the strength and resisting power of the patient. The height of the fever may be an index of the resisting power of the system, certainly some of the gravest and most serious conditions are those in which no rise of temperature occurs.

We have also reached the Socratic wisdom of admitting that we do not know what fever means, and are still in the dark as to the mechanism of its production, and the more intelligent no longer treat fever as such, any more than we are satisfied to prescribe for "dyspepsia," "general debility," or "muscular rheumatism." An enormous amount of thoughtful, painstaking and conscientious research has been done upon the problem, which, while it has not brought us to, or even apparently in sight of, the solution, has been exceedingly helpful in clearing away some of our misconceptions.

First, and most important among these was the hyperoxidation theory of fever. The idea that, to put it very crudely, the body metabolism had burst into a blaze, and the tissues were burning themselves up too rapidly, with consequent rise of temperature, as if the fire in the stove had spread to the walls of the house. This was a most attractive and plausible explanation, fitting in so beautifully with the flushed cheek, the burning skin, the throbbing temples, the sensations of heat and fulness, as well as the rapid loss of weight, the decline of muscular energy, the hot, quick breath, all of which seemed to show the nature of the trouble upon their very face. Was not the body an engine, did not heat drive the engine, and was not oxidation the source of that heat? A little excess of oxidation produces more heat, burns up more of the body fuel than is economical, a rise of temperature ultimately produces a wasted patient.

Moreover, our first tests with the thermometer showed that the temperature of an inflamed part, which was accessible to direct observation, was distinctly higher than that of the surrounding tissues. Nothing could have been clearer; but the moment that it began to be seriously considered, flaws appeared in it. The increased heat of the tissues at the seat of inflammation, for instance, was soon shown to be merely due to their having a more abundant blood supply pumped through them, with greater rapidity, from the depths of the great viscera. The flushed skin, the red eye of febrile reaction were shown to be due to precisely the same cause, the temperature, already in many cases, having fallen several degrees from the apparently cold and clammy stage of chill.

Careful tests with the calorimetric chamber showed that there was no appreciable increase in the total amount of heat given off by the body, in fact, in many cases, even a diminution. Last and most fatal accurate measurements of the amounts of oxygen taken in, and CO_2 given off, showed that, after a brief temporary increase, there was a return to nearly normal level, followed in continued fevers by an actual diminution in the amount of both intake and output of oxygen.

Moreover, the whole simile of the body as a steam engine, with heat as the motive power, is beginning to be regarded as untenable and misleading. The actual work of the body is probably done by the chemical or electric energy stored up in the complex food molecule, the heat produced is probably a friction waste-product, or an incident of "garbage burning." The body is *not* "burning itself up" in fever, our fever patients are *not* in the condition of a man doing heavy tasks of hard muscular labour, and all methods of treatment, based upon this conception, will have to be modified or abandoned.

Next, and closely connected with this, was the impression that the chief danger in fever was from the high temperature as such, which either directly burnt up and destroyed the body tissues at an excessive rate, or caused such changes in them as would result in their rapid breaking down and absorption. This is now believed to be unfounded. Experiments upon animals, placed under such conditions of heat, moisture, etc., as to raise the body temperature from 2 to 4 degrees, have shown that this elevation could be maintained for a considerable period of time, without producing any of the more serious symptoms of fever. A mere elevation of two or three degrees is not injurious to any healthy organism. The tissues of such animals were carefully examined after death, and, while not perfectly normal, were found to be entirely free from the familiar cloudy swelling of the protoplasm, which is so characteristic of true fever, if it is prolonged beyond a very moderate time. In other words, it has been pretty clearly established that the destructive changes in the tissues in fever are due, not to the elevation of temperature, *but to the toxin which causes the elevation.* Precisely similar changes may be brought about by the injection of toxins in large amounts *without the production of any elevation of temperature whatever;* on the contrary, in many cases, the temperature will actually be depressed below the normal (*Roger*).

In our own species, it has also been found that the actual temperature of the healthy human body is not by any means maintained at such a fixed and rigid level, as we had come to believe, but is capable of very considerable variations, under the influence of heat, of cold, of violent and prolonged exertion, etc., without any serious disturbance of the general health, or metabolism.

Perfectly healthy men at hard labour, or, in the tropics, may have a temperature of 100°—101° F. without any apparent injury. This points to the important practical conclusion, already glimpsed from the clinical point of view, that to merely reduce the temperature is by no means to cure the disease, or to necessarily improve the patient, that it is not the temperature, upon which we should fix our attention, and towards which we should direct our treatment, but the toxins of the infection which produce it.

Very instructive in this regard are the findings of Roger and Héricourt, who found that, in rabbits, the same toxin, which in moderate doses would produce a marked elevation of temperature, with the clinical symptoms of fever, in large doses would produce, instead of an elevation of temperature, a marked depression. If the dose were still larger, this depression of temperature would rapidly increase, and the animal die in frigid collapse within a few hours, showing clearly that an elevation of temperature is indicative of the possibility of resistance on the part of the tissues.

How many of our boasted and much-used febrifuges and antipyretics act simply like an increased dose of the toxin, by depressing the vital resistance and preventing the temperature reaction?

I have no hesitation in naming two, aconite and veratrum, and expressing grave suspicions of a third, namely, the whole group of coal-tar products. The man, who gives aconite or veratrum in a case of pneumonia, typhoid, or appendicitis, is pouring a second poison into the body of his unfortunate patient to suppress the resistance which it makes against the former. They make the patient more comfortable, and the doctor easier in his mind, for the time being, but what of the ultimate outcome? They lower the temperature, slow the pulse, but, much after the fashion that a blow on the head with a club will quiet the struggles of a rioter resisting arrest, or a dose of opium will relieve the fatigue of a soldier on the march.

Even the classic controversy, whose battle cries were "*Thermolytic*" and "*Thermogenetic*," has lost most of its interest and pertinency; for, if the accumulation of heat in the body in excess is not the cause of the distressing symptoms of fever, but merely one among them, and the real damage is done by the toxin, it boots little whether that increase in heat is due to an excessive production, as the "genetic" protagonists proclaim, or an inadequate dissipation, as their "lytic" opponents contend. *The heat does not do the harm*, nay, it is not even a reasonably constant accompaniment of harm done, or index of the amount of the same. So that it makes really no important difference whether it is produced faster than it can escape, or is prevented from escaping, as fast as it is normally produced.

The vital problems are those of *toxogenesis* and *toxolysis*. What influences produce, and promote the formation of, the toxin, and what influences neutralise and promote its elimination? So far as any decision has been arrived at, it would appear as if the thermolytics had a shade the best of the controversy, inasmuch as it has been clearly shown that there is no increase of oxidation, except for very brief periods, and these followed by a fall, and there is unquestionably, in fever, serious disturbance of that part of the vasomotor centre which is concerned with the dermal circulation and escape of heat. But to regard this disturbance of the so-called *thermotaxic* centre as the cause is little short of absurd, when it is obviously only a part of the general disorganisation and disturbance of metabolism set up throughout the entire body by the toxin, or toxins.

This heat-regulating mechanism is one of the wonders of the animal body, so exquisitely responsive, so elastic, so resourceful, that it would take a whole chapter to describe even the barest outline of the more important feats of which it is capable. Physiologists are never weary of singing its praises, and, from our student days, we have been continuously called upon to bow down and admire its excellences, until we have unconsciously adopted an attitude of reverence towards it, which has not exactly promoted a rational understanding of its variations. We have come to speak of it as if it possessed an independent intelligence of its own, and devoted its entire effort to the maintenance unchanged, in all circumstances, of that sacred and never-to-be-departed-from standard, 98·6° Fahrenheit. Moreover, so great is its constancy, and so unerring the certainty with which it will return to this particular level under all sorts of variations of environment, that we have been led to regard this particular pitch of temperature as one of the most vital of vital requirements for the welfare of the body, as an absolutely indispensable requirement in the physical plan of salvation.

I believe that we have here been again misled, and this brings us to the particular questions, which I wish to raise, and in regard to which this paper is simply to be regarded as an enlarged interrogation point, namely, the meaning and importance of temperature, both normal and abnormal, in the animal body.

A very brief examination of this remarkable heat-regulating mechanism shows us that it is by no means so uniform, or so exquisitely rigid, as we have been in the habit of assuming.

In the first place, it is, as Burdon-Sanderson acutely remarked some years ago, a process which is chiefly concerned with providing for the *escape* of a certain amount of heat, and not with its production. Heat is constantly produced in excess of any normal utility which it may have in the body. If its escape were not provided for, discomfort and ultimate disturbance of metabolism would probably occur, consequently the piling up, to a certain pitch, of the waste products of metabolism, associated with the liberation of heat in the body, acts as a stimulus to the vasomotor centre in the medulla to dilate the superficial blood-vessels, to increase the force and rapidity of the heart beat, and either the depth or frequency of the respirations, thus exposing large amounts of blood to the external air in the skin and lungs, and permitting the escape of its heat. A rise of external temperature produces something of the same effect in less degree, with the added effect of the pouring out of perspiration and increase of loss of heat by evaporation.

There appears to be no mechanism needed for providing against the contingency of the heat being produced in deficiency of the needs of the body, for the reason that this, in conditions of health, and, even within the range of a considerable departure therefrom, is practically never known to occur. The few cases, in which it does occur, are those to which we have already alluded, of profound intoxications, which rapidly prove fatal, and hence cannot leave any after-impress upon the organism or race.

No adequate proof has been given by any investigator of the existence of a true thermogenetic mechanism, or centre, capable of either diminishing, or increasing, the actual production of heat in the body ; so that all that is really required in this boasted mechanism, is a condition of the blood vessels, whereby they relax and expand under heat, and contract and and diminish in size under cold. A power, which it need scarcely be pointed out, is possessed by a simple inorganic metal like iron. That this power has been transferred to a so-called centre with power to regulate, control, and balance it in various parts of the body renders it no whit more remarkable, or different in degree. We have been altogether too fond of imagining that, when we have discovered a localisation centre for some particular action or function, we have reached a final explanation of it. Whereas all we have actually done is to shift the problem one step backward. All that a centre can possible do is to *regulate* an activity, whether a motion or a secretion ; it can *originate* nothing. Like all other rulers, it derives its power from the consent of the governed.

Even this degree of heat-regulation is open to a curious suspicion as to whether or no it is primarily for this purpose at all. As can be seen at a second glance, the whole of these changes of superficial flushing and paling are accompanied by an entirely different and much more important process, namely, the escape of waste. When the capillaries of the skin-mesh and the lung-mesh are distended, and greater quantities of blood are pumped through them, not merely is heat escaping, but poisonous excretory matters, as well, in the one case, large amounts of CO_2 and smaller amounts of poisonous nitrogenous and fatty extractives, and in the other, considerable quantities of the latter with smaller amounts of the former. Perspiration, with its alleged cooling effect, is accompanied by an even more striking amount of escape of poisonous waste materials. In fact, almost the whole of these alleged heat-escape changes could be explained in terms of waste-escape.

This is perfectly clear as regards increase of heat dissipation, but not so clear as regards decrease due to a lowering of the surrounding temperature. This latter is certainly not attended by any necessary diminution of the amount of waste which is to be got rid of, and yet there results far less activity of the skin. But here promptly come in two other mechanisms. There are two other organs, or regions, in which increase of activity almost invariably accompanies decrease on the part of the skin, viz., the lungs and the kidneys. Exposure to cold air is well known to increase the amount of nitrogenous excreta given off by the lungs, while, at the same time, the temperature of the expired air is raised slightly, and similar action takes place in the kidneys. Chilling and paling of the skin by exposure to cold air is notoriously accompanied by increase in the amount of urine, and in the output of urea and other nitrogenous excreta. The whole problem may be one of the elimination of toxins. Are we not coming to the same point of view in regard to fever?

This fixed temperature of our bodies is subject to much wider and more frequent variations than we usually recognise. Not only does the temperature vary through a range of nearly a degree, according to the situation in which it is taken, the mouth, the axilla, the rectum, thus raising a suspicion that it is a question of the abundance in which hot blood from the interior is thrown out to the surface, which is one of the chief factors in a so-called rise of body-temperature, but there are marked fluctuations in this superficial temperature in health, and in various conditions, which are not necessarily associated with disease.

For instance, the sacred 98·6° or 98·4°, instead of being a fixed thing, is simply an average of the daily temperature of the normal individual taken between the hours of 10 a.m. and and 4 p.m. When we begin to extend our researches to the earlier hours of the morning and the late hours of the afternoon, differences are shown. Our normal patient will register some other figure at least three times as often as he will register just precisely 98·6°. In other words there is a regular diurnal variation of temperature corresponding with the degree of activity, both muscular and metabolic, carried out in the body, extending over a daily range of a degree to a degree and a half Fahrenheit, lowest in the early morning, and highest in the hours between mid afternoon and early evening.

If a certain temperature is an absolutely indispensable factor in the normal environment of healthy tissue, then our bodies are in distinctly abnormal conditions, in the early hours of the morning, and again in the late hours of the afternoon and early evening. Again the suspicion is raised that temperature, or vital heat, as we have termed it, is merely *an incidental function of the metabolism of the body and not a vital necessity.*

Further than this, there are of course also changes between youth and old age ; the temperature of the child being usually given as slightly higher than that of the adult, and that of the old man the same. Authorities vary somewhat in their findings in this respect, and it seems most probable that the temperature, instead of being absolutely higher, or absolutely lower, at either of those two extremes, is slightly more changeable, and covers a wider daily range. The heat-regulating mechanism is certainly imperfectly developed in children. Further than this, careful experimentation has shown that the temperature of the body, particularly when deeper parts like the rectum, or the interior of large muscles, or of the great viscera, are taken as tests in animals, may undergo appreciable and even considerable variations, in various circumstances, without any disturbance of the general health being discoverable. For instance, under vigorous and violent muscular effort, there is distinctly appreciable rise of temperature in perfectly healthy individuals. Exposure to high external temperature will also be followed by a rise of from a half to three-fourths of a degree, and even a disturbance like hysteria, or sudden shock, without ascertainable organic lesion, will be attended by marked rise of temperature, yet with little or no disturbance of the general health, and no suspicion of the other phenomena of fever.

We are now coming to realise, from extending our temperature-taking over 18 hours out of the 24, instead of 8, that the essence of fever is not an absolute rise, not yet an absolute fall, but *an increase in the range of daily variation.* This increase is simply an exaggeration of the normal tidal rise and fall that takes place in perfect health. In other words, our conception of fever should be revised from a mere elevation above a certain fixed level, say 100° Fahrenheit, to an increase in the normal daily variation in excess of a degree and a half. A perfectly normal individual, for instance, will range from 98·2° in the morning to 99° in the afternoon, a variation ef eight-tenths of a degree. A typhoid case, on the other hand, will run from 100·5° in the morning to 103° in the afternoon, a range of 2½°; a tuberculous case will range from 97° in the morning, to 101·5° in the afternoon. The chief element in fever is the marked increase in the daily range of temperature. Clinically, our definition of fever should, in my judgment, be *a daily range of temperature in excess of one and a half degrees.*

It may be nothing more than a coincidence, but we have revised our conception of warm-blooded and cold-blooded animals on almost precisely the same basis. The warm-blooded we call *homoiothermal*, or "even-temperatured," by that really meaning a daily range of temperature of only a very limited extent. The cold-blooded we now term *poikilothermal*, or "changeable-temperatured," because their temperature has so much wider range. They are generally actually colder-blooded than the homoiothermal forms, because the average temperature of air, or water, is below that of mammalian blood, but in a sufficiently warm medium, either aerial or aquatic, their temperature may rise to mammalian, or even fever heat, as shown by Sutton's observations of a temperature of 103° in a python lying in the sun.

This brings us to the crux of the whole problem, upon our answer to which both our conception and treatment of fever will depend, and that is what is really meant by body temperature. Is it a vital necessity, or a mere accompaniment of vital processes, which may or may not be present ? There might be some doubt as to our answer to the former part of this question, so long as we confine our view to mammals and birds, in all of whom a fixed body-temperature of a certain degree of elevation is a constant condition ; but the moment we look outside of this restricted field, the question answers itself at once, for we see cold-blooded animals, with body temperatures only removed a few degrees from that of the sea water, or air in which they live, carrying out absolutely every activity of which our bodies are capable, only in less degree.

The whole world of plants is capable of a constructive metabolism and growth, which our bodies can only feebly imitate to a very limited extent, without even a fraction of a degree of elevation of temperature above that of the surrounding earth, air, or water, which may be within a few degrees of the freezing point. Growth, movement, reproduction, constructive metabolism of the most elaborate sort are all alike possible in the absence of heat. Of course I do not mean absence of heat in the sense of absolute zero, which point as yet has never been reached, but only that vigorous and elaborate metabolic and vital activities can be carried on at a temperature far below that which we are accustomed to regard as indispensable to life. Energy of some kind, of course, must be used up in all phenomena, chemical or vital, but that energy is probably much more frequently electrical or chemical in form, than thermal. In other words, although vital activities are carried out better in the presence of a moderate degree of heat, it is doubtful whether the actual work done is by thermal energy, and whether the heat accompanying this process, like that given off by the electric light or fan, is not, to a large degree, a waste product. Plants furnish us a beautiful illustration of this, inasmuch as they may possess a temperature not only no higher, but actually lower, than that of the surrounding air. When they are engaged in rapid growth, or starch storage, the synthetic processes may be so much in excess of the analytic as to cause absolute absorption of heat instead of liberation. In so far as they form waste, as it is now known that they do, in the shape of minute quantities of CO_2, they probably liberate heat, but it is in such exceedingly small proportion to the bulk of their total activities, that the net result is a reduction of temperature instead of an elevation.

Of course most plants thrive better in the presence of moderate heat, but the question which I wish to raise is, whether this is not chiefly a favouring condition for the interchange and play of the real life—energy, electric, chemical, or photic—rather than the vital force itself. Certainly plant growth can be powerfully stimulated by electric light or currents, which give off no heat at all, and no matter how warm plants are kept, they grow very poorly in the absence of light. It is the light of the sun, rather than its heat, which acts upon the chlorophyll granules to form starch.

The researches of Davenport have shown that, in certain abnormal circumstances, for instance in the repair of a wound in their tissues, plants will exhibit a slight but traceable rise of temperature, in other words, a sort of vegetable fever: but this would appear to be due to the increase of destructive metabolism, set up by the wound, or involved in the process of repair.

Any one, who in Alpine rambles has seen the little *Schneerosen* pushing their heads right up through the snow-crust, or who, climbing higher, has seen the "red snow," due to a powdering over of its surface with swarms of tiny fungi, thriving and reproducing upon the frozen crust, will have no difficulty in dissociating vital activity from heat.

The majority even of plants will, of course, flourish better in a moderate amount of heat, and an excessive amount is just as injurious as a deficiency. The same is true of light, electrical tension, moisture, nitrites, and a number of other factors in the environment. Heat is a useful factor in plant growth, but is it anything like as indispensable as we have usually assumed?

The case of the "cold-blooded" animals is somewhat different, though parallel to this extent, that they depend largely upon the surrounding medium for the precise pitch of their body heat. But their temperature, while it is almost that of the surrounding medium, is never quite identical with it, and they have limits both above and below which vital activities cease. As before intimated, we use the term "cold-blooded" simply as one of convenience, and because it is already widely known, and clearly defines the class of animals to which we refer. It is, however, a misnomer, because animals of this class, though cold-blooded in a cold medium, are warm-blooded in a warm medium; and further because, while their temperature varies with the surrounding medium, it is never exactly identical with it. The frog, for instance, living and moving actively in water at a temperature of 66° F., shows a stomach temperature of 0·5° higher. If that water is raised to 75° or 80°, he will only follow the upward change with his temperature to the extent of 5° or 6°, say to 70° or 72°. If, on the other hand, the water is chilled down to 48° or 50°, his temperature will drop to about 55°. In other words, he has a sort of central norm around which his temperature variations circle, keeping between it and that of the surrounding medium.

This is even more clearly seen when the frog is out of the water, for here his temperature with the surrounding air at 5·9° C. will be 18·6° C., and with the air at 40·4° C., his stomach temperature will be 31·7° C., a "reserve" of 23° F. at one extreme, and 15° F. at the other. As is well known, when the temperature of a cold-blooded animal is reduced below about 40°, vital activities are practically suspended, and the animal passes into a stage of so-called suspended animation or hibernation, in which he will remain for almost indefinite periods until the temperature is again raised.

It must be remembered that this apparently utterly uncontrollable and irregular range of temperature is, in ordinary circumstances, much less wide than we might at first sight imagine. The large majority of cold-blooded animals are aquatic, or subterranean in their habits, and the temperature of either large bodies of water or of small ones, if these are in constant motion, seldom falls below 55°, or rises above 70°. The total range of variation in cold-blooded animals falling from 2° to 4° inside of these limits at either end, will not exceed 10° or 12°, and often no more than 6° or 8°. Moreover, these changes in water are, as a rule, made very slowly, so that the actual temperature of a given poikilothermal animal in ocean, lake, or river, may be more constant, for months at a stretch, than that of his so-called constant temperatured, or homoiothermal kinsmen on dry land.

The differences then between the mechanism of cold-blooded animals and of warm-blooded animals are not so striking as supposed, and are evidently only in degree instead of in kind. The cold-blooded animal has less power of controlling his bodily temperature and a wider range, the variations of which, however, take place very slowly, and over much wider intervals of time. The heat-producing processes, whatever they may be, in his body are less than in our own, but they are apparently even more constant for weekly, or monthly periods than ours are. Our temperature varies in daily excursions over a narrow range, his in monthly, seasonal, or semi-annual, over a wider.

The curve of our temperature is like that of the contraction of striated muscle, his like that of nonstriated. Drawing a definite norm through the centre of the variations for a year, our oscillations away from that medium would be found to exceed his. We vary a $\frac{1}{2}$° from that base-line twice a day, he 5° four times a year. Yet he is just as emphatically alive as we are, and capable of every possible form of activity of which we are capable. From the standpoint of absolute vital accomplishment, a worm is just as good in kind as a man.

What then seems to be the chief distinction between the cold-blooded and the warm-blooded animals? Mainly that the one is capable of carrying out his vital activities with less waste of energy in the form of heat than the other. Both alike derive their energy from the potential energy contained in the complex molecules of their food, one transforming that potential into vital work with less friction waste in the form of heat than the other.

Would it not seem, in other words, that the cold-blooded animal is a more economic and less expensive machine than the warm-blooded? This, I think, is distinctly indicated by the amounts of food consumed. Anyone who has had to do with the feeding of frogs, fish, or snakes, as compared with mice, rats, and guinea-pigs, of nearly the same weight, will have been struck with the astonishing discrepancy between the amounts of food required in the two cases. A two-ounce gold fish, even when kept in a large aquarium, and allowed opportunity for all the exercise he wishes, is well fed on a few scraps of wafer, or crumbs of bread, not exceeding 15 or 20 grains weight per diem. A mouse of the same weight kept in the tinniest of tiny cages, and sleeping the greater part of the time, will eat five times that amount. The principal part of the difference, of course, is accounted for by the amount of muscular work done by the two animals, as measured by the amount of movement carried out. While the fish, as a machine, is just as complicated and elaborate as the mouse, the latter is geared to a higher rate of speed tension, and is, of course, in consequence, capable of filling a different sort of a gap in the interstices of the universe than the former.

In other words, the difference between the two machines is that one of them is absolutely limited to a certain rather rigid, but very common group of surrounding conditions, while the other has adapted itself to a different set of conditions, less common in the universe, but of wider range of variation. As Romanes acutely pointed out, the struggle for existence is only in part a tooth-and-nail struggle for the possession of certain conditions and certain food, and is also a struggle for qualities, which will enable the individual to occupy areas, or "gaps," in the environment, which are not suitable or viable for other forms.

The specialties of the cold-blooded animals are, to put it very roughly, the domain of the water and the earth below the surface, where neither birds nor mammals can compete with them to advantage. They have developed a capacity for living upon a minimum of food, with a minimum of waste of energy in the form of motion and consequent heat. The warm-blooded animals, on the other hand, having worked their way up to the surface of the dry land, and adapted themselves more perfectly to this *milieu*, have acquired the habit of much more rapid and frequent motion. This has involved a greater waste of energy in the form of heat, and a corresponding elevation and constancy of temperature. This very constancy has enabled them to further extend the sphere and constancy of their operations, and this, of course, again means still further and more persistent heat-formation.

It has been clearly demonstrated by recent observers that the cold-blooded habit, possessed by so many mammals, of hibernating during the winter time, in other words, sinking, for the time being, back to the ordinary cold-blooded level, is due not so much to intensity of cold as to scarcity of food. For instance, scarcely any pure carnivora hibernate, because a certain amount of supply of their food is to be found the whole winter through. Foxes, wolves, weasels, minks, martins, etc., are as active in winter as in summer. The same is true of the herbivora, who are capable of living upon dried or frozen grass, leaves, branches, and other materials, which are accessible in the winter time; the whole deer family, the bison, and the browsing group generally making no attempt whatever at hibernation. Only those herbivora, in fact, which depend chiefly upon seeds, nuts, berries, roots, which are absolutely unobtainable during the winter, like the squirrel, rat, and mice families, the porcupine, the woodchuck, and the bear (the last, in spite of his bloodthirsty reputation, is very largely a nut, grain, and berry feeder) undergo this process. Many of these animals, when kept in captivity, and supplied with an abundance of food, show little or no tendency to hibernation. In other words, when the warm-blooded animal can no longer get the food fuel, which is necessary to supply his persistent waste of energy, and consequent formation of heat by bodily movement, he drops down to the slow minimum activity of the cold-blooded animal, and economises energy by suspending motion, including even that of active respiration, during the period of scarcity, until it has passed. If only some of us highest mammals had managed to retain this faculty, what a precious resource it would be!

Birds, with their high temperatures and incessant activities, never hibernate, but solve the problem by migration, except a few hardy flesh-, insect-, and seed-eaters.

That this is the state of affairs is also strongly suggested by the proportional oxygen intake in cold-blooded, and in different forms of warm-blooded animals. The worm, for instance, will carry out all our vital activities with an absorption of one and one-half grams of oxygen to every gram of his body weight per diem. The frog, with no higher degree of metabolic, but much higher motile activity, requires from 2½ to 3½ grams; the human species require from 9 to 12 grams; the dog 12 to 15, and birds 25 to 35 grams. The body temperature rises along the same scale, higher in the frog than in the worm, higher in the bird than in the mammal.

But some one will say at once, of course the oxygen intake is in proportion to the amount of work performed, and is not this excess of work due to the combustion which this increased oxygen intake makes possible? In other words, is this not simply a gauge of the draft of the body-furnace under the vital engine? Some recent researches of Loeb, upon the rôle of oxygen in cold-blooded animals, throw a most interesting light upon this point. By a series of ingenious and elaborate experiments, whose conditions were so skilfully varied that possibility of error would seem to have been almost absolutely excluded, he came clearly to the conclusion that the oxygen inhaled by cold-blooded animals is not used in carrying out the actual vital processes in the cells themselves, but in oxidising and neutralising certain poisonous waste products, formed in the course of these vital processes, and which would inhibit their further continuance if not so neutralised. Work could be kept up, in muscles entirely deprived of oxygen, for a considerable length of time, with the result that the accumulation of waste products of motor metabolism would finally reach such a point as to inhibit further activity. Then the introduction of oxygenated blood into the vessel supplying the muscle would change these to CO_2 and other comparatively non-toxic waste products, and activity would be promptly resumed.

Is it not highly probable that this is the rôle that oxygen plays in warm-blooded animals, not as a factor in combustion with consequent production of heat and energy from that heat, but simply as an oxidiser and neutraliser of the poisonous products of actual vital metabolism? In other words, is not the heat formed in our bodies chiefly due to a process of waste destruction, a sort of garbage burning?

A brief glance at the temperatures of different warm-blooded animals is of interest in this connection. If body heat is an absolute vital necessity, then each particular animal has found a different degree of it necessary to its welfare. Beginning with the lower and more sluggish forms among the marsupials, with a temperature as low as 86° or 87° F., it runs the gamut of the animal kingdom, up to 118° in certain birds. In other words warm-blooded (*homoiothermal*) animals, as a class, have nearly trebled the range of any single cold-blooded (*poikilothermal*) animal. Each species would have seemed to have settled this vital problem for itself, our own species occupying a relatively median position.

Below us are the marsupials and certain of the lower forms of birds, like the apteryx. Our most intimate associates, the cow and the horse, have a nearly identical standard, 99°—100°, while the dog and the rat are both above us, with a temperature of 102°; the goat and the sheep agree upon the same level, namely 104°; the pig comes up to 105°, while the rabbit asserts a curious individuality, varying from 100° to 105°. Birds, for the most part, have a considerably higher temperature than our own, ranging from 105° to 110°; the swallow and one or two other exceeding active forms reaching as high as 118°.

Does not this variability look more like a function, which was merely a working equation between growth and waste, between nutrition and motion, in the mechanism of each particular species, rather than an absolute vital necessity? In any of these forms, disturb this balance by the introduction of some toxic irritant, and you increase the range of daily vibration of the equilibrium between the processes of growth and those of waste, with the resulting production of what we term fever.

I am afraid that this will seem rather a wide digression from the bedside, and the practical problems of clinical medicine. But I believe that it will be more than justified, by the increased breadth of grasp and accuracy of view, which it will give to our conception and consequent treatment of fever.

Take the hypothesis into the sick-room, and test its application to the clinical facts and effects of fever. In the first place, it agrees precisely with the fact, upon which practically all investigators are now agreed, that the one change, which is absolutely constant in fever, is an increase in the output of nitrogen ; some of it in the form of urea, more in that of uric acid, or other nitrogenous excreta. This means an increase of destructive metabolism in the tissues themselves, with the result that the energy, derived from food, stored up in them, instead of being liberated in the relatively cool, smooth, and frictionless form of growth or secretion, is wasted in the form of heat. Fever is always pathological, not in any sense an increase of the normal work of the body, with a consequent increase of temperature, such as would be produced in a steam engine by increasing the combustion under its boiler.

The next thing, with which fever is almost invariably associated, is an impairment of function, almost to the degree of stoppage. The higher the temperature, the more completely the body is incapacitated. Bile, gastric juice, saliva, even perspiration, cease to be secreted, muscular activity sinks to the lowest level, and all the activities of the body become clogged and impaired. There is an almost utter stoppage of all useful metabolism, with sudden conversion of the energy normally used in this, into the waste product—heat. Further than this, it explains how, if the process goes a step further, by utter failure of the powers of resistance, or from excessive dosage of the toxin, this stoppage of metabolism becomes so absolute that not even enough waste in the form of heat is produced to keep up the body temperature. This latter rapidly fails, the patient goes into a collapse, and, if the process is not speedily checked, dies. Under the influence of moderate doses of toxin, the bodily engine is, to use a very rough simile, breaking itself to pieces, grinding out its bearings, producing friction instead of performing its natural and necessary work. Let the dose be excessive, and it may absolutely stop activity of every sort.

It also makes clear why there is not necessarily a constant increase of oxygen intake, and CO_2 output in fever, inasmuch as the metabolism, which is going on, instead of being in *excess* of the normal, is really *below* it. Instead of going on through to its normal end-products, which can be readily oxidised by the oxy-hæmoglobin, and given off as the harmless waste product carbonic acid, under the disturbing influences of the infection, chiefly half-way products are formed, and smaller amounts of this readily oxidisable material, the remainder of the energy being liberated in the form of useless heat. During vigorous and prolonged muscular activity in healthly individuals, there is a marked increase in the amount both of oxygen intake and CO_2 output. But this is promptly met by an increase in respiratory and cutaneous activity, and relatively harmless waste products rapidly escape from the system, and, though here is a slight rise of temperature, there are none of the phenomena of fever. Not an *excess* of metabolism, but an *abnormal form* of it is the secret of fever.

It is also in agreement with the attitude toward, and treatment of fever, to which our clinical experience has already led us, so trite as to make it almost superfluous to allude to it. If the increase of temperature is merely an indication of the amount of disturbance of the body mechanism by the infection, and very largely of the amount and quality of the toxin, whether of *exo* or *endo* origin, we see at once why it is worse than useless to endeavour to treat the rise of temperature as such. We are perfectly justified in regarding it simply as an indication, a symptom of a toxic process (a symptom which, by the way, may or may not be present, according to the character and degree of the intoxication), and in concentrating our attention solely upon the discovery, and, if possible, the neutralisation or elimination of this toxin.

We should never forget that a *depression* of temperature may be just as genuinely a sign of intoxication as an *elevation*, although it will less frequently occur. In certain infections like cholera and consumption, and morbid states like diabetes and uræmia, it is nearly always to be observed, if looked for. Our conceptions of both intoxication and infection need broadening.

This view-point also helps us to see more clearly how a considerable part of what we are pleased to term a rise of temperature, though a part of it, and often the bulk of it, may be due to an actual increase in the amount of heat generated in the tissues, lies in the larger amounts of the heated blood of the internal organs thrown out towards the surface of the body.

The same parallelism obtains here that does in normal temperature regulation; it is most closely associated with an attempt at elimination of waste products. In fever, the amount of waste in the blood is raised to abnormal pitch; consequently there is a desperate attempt on the part of the heart to drive it out to the surface, both cutaneous and pulmonary, for purposes of elimination. Part of the increase of heat, both of the surface and of the expired air, is undoubtedly to be accounted for in this way. At the same time, by one of the *circuli vitiosi*, so familiar to us, the very toxins, which so stimulate the heart, will promptly paralyse both the general metabolism and that of the sweat glands, the respiratory epithelium, and the glomeruli, and tubules. It will even produce a contraction of the calibre of the superficial vessels, so that the skin, in the late stage of fever, becomes pale and shrunken, instead of being red and full. Any procedure, therefore, which will tend to relieve the surface conditions, restoring the tone of the cutaneous blood-vessels, and arresting the paralysis of the sweat glands and pulmonary alveoli, will be of value in assisting nature to get rid of the infectious materials. It has long been obvious that baths, in typhoid and pneumonia, and packs, in scarlet fever and measles, act in a far more potent and radical way then merely by lowering the temperature. It is by deepening the respirations, steadying the heart, increasing the flow of urine, and restoring the pulsations of the great skin-heart, that they do good in fever, in a far higher degree than by a mere increase of the escape of heat.

And to give even the devil his due, it is possible that the coal-tar products produce some of their antipyretic and relieving effects by promoting perspiration, and mucous secretion, with their accompanying elimination, though probably more by either obtunding the cells to the effect of the toxins, or by lessening reaction by depressing resisting power. They are unrivalled analgesics, fever opiates, so to speak, without any of the disturbing, secretion-clogging after-effects of opium, and, if regarded and given *solely as such*, have a distinct and rather diversified field of usefulness. Always to be regarded as depressants, and watched jealously for heart-effects, in mild febrile attacks, or in vigorous individuals, where the patient has, so to speak, plenty of resisting power to spare, they certainly do make him much more comfortable during the process of reaction ; but in continued sieges, where every ounce of resisting power is going to be needed, they are not only useless but harmful.

To sum up, the following tentative conclusions in regard to fever seem justifiable for heuristic purposes :—

(1) That it is not the rise of temperature that does harm, but the toxins present.

(2) This rise of temperature is not due to increased oxidation, as shown by normal or lowered CO_2 output.

(3) The phenomena of fever appear to be due to a general disorganisation and perversion of normal metabolism by toxins, with conversion of the energy, normally expended in secretion, growth, motion, etc., into the waste-product heat. To use a crude simile, it is a "hot-box" all over the body-engine, as the engine may be regarded as having broken a shaft, and grinding itself to pieces with evolution of heat from the abnormal friction.

(4) There is actually less metabolism going on in the body in fever than in health, but it is chiefly destructive instead of constructive.

(5) If the dose of toxin is sufficiently large or virulent, this lessening of metabolism may go so far as to lower the temperature.

(6) The temperature in "fever" is often subnormal.

(7) Our "standard" of fever should be *an increase in the daily range of temperature in excess of* 1·5° *F.*

(8) The rise of temperature in fever may possibly be secondarily protective, as many pathogenic organisms are unfavourably affected by exposure to fever temperatures. This effect is doubtful and slight at best.

(9) Normal body-heat may be a friction-remainder, a waste product turned to use, rather than a vital necessity.

(10) Treat the intoxication, not the temperature.

JUVENILE GENERAL PARALYSIS.

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THE subject of this paper may perhaps, at first sight, seem to appeal to only a small section of physicians, and to be more suited for the consideration of the alienist than for that of the medical profession in general. I feel it necessary, therefore, to attempt in a few words to justify its selection. I do not think that there are many of us who do not in practice, from time to time, encounter the mentally defective child; and one of the most pressing problems in dealing with such cases relates to the prognosis. It is, therefore, of the most vital interest to the practitioner to be in a position to recognize any form of abnormality which may involve a definite forecast, and such a condition is juvenile general paralysis, in which the outlook is invariably fatal. Again, the disease, though not common, is by no means one of the rarest forms of mental breakdown in childhood, and its incidence appears to be on the increase. For the purpose of this contribution, I have analysed the records of over twenty of the cases which have come under my personal observation; in eleven of these a post-mortem examination was made. I am inclined to place the percentage of general paralytics, in any series of imbecile children, at about five. Owing to its fatal termination, the condition is met with in greater proportional frequency at autopsy, and, in the records of 18 months at a large imbecile institution, I have found the percentage at post-mortem as high as seventeen. I trust that these considerations may be held to qualify my subject for a claim on general interest.

The affection may be defined as a condition of progressive deterioration of mind and body, the outcome of stress operating on a nervous system tainted with hereditary syphilis, and marked by certain mental and physical characteristics. It must be clearly recognized that a congenital imbecile may, and not infrequently does, become the victim of general paralysis in this form, and, in such a case, the mental history may be obscure.

In the majority of instances, however, the subject is a child, who has shown good, or average, intelligence, until the supervention of one or more exciting causes in the form of stress have strained, to breaking point, the insufficient resisting powers of his central nervous system, and initiated a premature decay, much as an exertion will overdraw the balance of a diseased heart. The determining factor is not always easy to trace; it may be the strain of school life, the onset of puberty, the necessity of making a living, or a variety of other causes. But, in all cases, the statement that mental symptoms are progressive should put us on our guard.

The connection with parental syphilis is fairly closely defined. I have never met with a case from which it could be excluded, although the familiar stigmata are not infrequently absent. It should be remarked that, in some cases in which signs of congenital syphilis are absent in the patient, they may be seen in brothers or sisters who are mentally sound. In other instances, careful inquiry from the friends will supply the necessary evidence of taint. In the case of one child, who showed the typical features of the disease, no point suggestive of syphilis could be elicited from the family, until it was discovered that the mother was attending Guy's Hospital for tabes dorsalis. In another instance, the father, who stoutly denied the possibility of venereal infection, proved himself to be a well-marked case of tabes with optic atrophy.

The age of onset is given by Mott, in his analysis of 22 patients in the London County Asylum, as averaging 17. As my own experience of the disease has been almost entirely among children, the average age in my series would be a great deal younger. In a case which I showed at a meeting of the Clinical Society in 1902, the symptoms had commenced at eight and a half, but I have since had a child under my care, in whom signs were well defined at seven and a half, and who has subsequently shown, at post-mortem, the characteristic lesions of general paralysis.

To turn to the clinical phenomena, the cardinal physical signs may be enumerated as iridoplegia, affection of speech, alteration of deep reflexes, and muscular tremors. The first is generally observable early, and is frequently suggested by inequality and irregularity of the pupils. Sluggishness or absence of light reflex is the commonest condition. Slovenliness of speech, progressively deteriorating, is fairly constant in cases seen sufficiently early, and a similar change may be noted in the handwriting. The knee jerks are usually exaggerated early in the course, and lost later. Coarse antero-posterior tremor of the tongue I have noted as an early symptom ; later, fine tremors of the facial muscles, especially on voluntary movement, and sometimes also of the hands, supervene in many instances. I have come to regard a curious facial expression of preoccupation and worry as typical of the earlier stages of the disease. It contrasts, very distinctly, with the cheerful unconcern or blank indifference of the ordinary imbecile, and with the leaden torpor of the epileptic. I can only recall one facies which at all suggested the condition of *bien-être* associated with the acquired disease.

Definite delusions are far less common than in ordinary general paralysis, and megalomania is rare, at least in children. Periods of acute excitement or depression are occasionally met with, but, in the majority of instances, the mental record is one of dementia of indefinite onset, unbroken by maniacal or melancholic accesses.

As the disease makes headway, diagnosis increases in difficulty. Physical weakness becomes more marked, until the sufferer is bedridden, with contracted limbs. The face loses expression—this is sometimes an earlier symptom—speech is lost, the deep reflexes are abolished, emaciation becomes more profound, until the condition is the exact counterpart of the last stage of the acquired form, death occurring from bedsores, or from some intercurrent ailment. Facial tremor often persists throughout, and the purposeless movements of lip and jaw associated with basic meningitis are common in the last stage. Epileptiform seizures may be present at any period.

Post-mortem, the brain presents the typical picture of a chronic diffuse meningo-encephalitis ; thickening, opacity, and adhesion of the pia-arachnoid, wasting of convolutions, especially in the frontal and central regions, increase of subarachnoid fluid, narrowing of cortex, dilatation of ventricles, and granularity of ependyma, the last usually most definite in the fourth ventricle. The lesions are thus identical with those of the adult form.

I now proceed to sketch briefly some illustrative cases :—

1. Lily L., aged 12, first observed, October 24, 1903. Went to school at four and a half, was very bright and quick, but slept badly, and used to repeat her lessons while asleep. No stigmata of congenital syphilis, but her mother has tabes dorsalis. A thin child, with characteristic look of distraction. Faulty in habits. Cannot walk. Right pupil larger than left; both are irregular, and do not respond to light. Knee jerks much exaggerated. Overaction of right orbicularis oris in speaking.

April 22, 1904. She is becoming feebler, and has developed the typical slurring speech of the general paralytic. This girl is still alive. In personal and family history, and in physical signs, she satisfies all requirements for diagnosis.

In the next case, the physical signs and progress afforded sufficient data for a diagnosis, which was confirmed after death.

2. Alfred W., aged 11½, first observed, January 15, 1903.

No history, family or personal, available. No signs of congenital syphilis. An undersized boy of fair nutrition; intelligence very limited. Both pupils polygonal; left larger than right, and not reacting to light; right shows sluggish light reflex. Knee jerks exaggerated.

February 3. Fine lip-tremor and coarse tongue-tremor noted.

June 18. Speech has become typically slurring. Patient getting weaker.

August 11. Death from tuberculosis. Post-mortem, the usual lesions of general paralysis.

The succeeding instance typifies the disease as seen in the congenitally backward child.

3. Florence I., aged 12, first noted, March, 1903.

Was late in learning to walk and speak, but went to a "defective" school for five years. Deterioration first observed a year ago. She has notched incisors, rhagades, depressed bridge, and antero-posterior bowing of tibiæ.

A restless, unhappy-looking child; intelligence very small. Left pupil is the larger, and reacts little or not at all to light. Right reacts normally. Knee jerks much exaggerated. No tremors. Can get about.

April 21. Excited, noisy, and sleepless, screaming constantly at night. This acutely maniacal condition persisted for some weeks.

Oct. 14. Child getting feebler. Unable to walk now.

Dec. 29. Died of pneumonia. Post-mortem, brain condition typical.

The last case, I shall quote, is of interest as apparently dating from an early encounter with the world, and for the supervention of delusions.

4. Lily H., aged 15, admitted to Darenth Asylum Schools, January 13, 1902.

She had passed the seventh standard and gone into service as a nursemaid, but was sent to Cane Hill in 1901, suffering from depression, supposed to have been caused by a fright. When seen at Darenth, she was apparently a girl of average intelligence for her social station, bright and pleasant looking; the medical officer's diagnosis lay between juvenile general paralysis and convalescence from melancholia. She soon proved to be forgetful and slow in her ways. My attention was called to her, in July 1903, by a sudden attack of transient blindness. On examination I found her right pupil larger than the left and immobile; her tongue was tremulous and her speech slovenly. Five days later, a similar attack of blindness occurred, with weakness of the right arm and increased right knee jerk clearly apparent, while the girl was for a time unclean in habits. In the early part of January 1904, subacute mania intervened, with marked delusions that her friends and those about her were dead, that she was going to be married, and ocasionally that she was a queen. Her habits were wet and dirty. This attack passed off after about two months. I hear that the patient died at Darenth last May; unfortunately no post-mortem examination was made.

This, the oldest of my cases, which I have had the opportunity of observing in an early stage, is also the only one in which delusions of grandeur were recorded, and it is worthy of note that in Mott's series, in which the average age was much older, megalomania was more frequently met with, although less common than in the adult form.

In conclusion, I would refer those interested in the subject to the article by Mott, in the first volume of the *Claybury Archives of Neurology*, which marks a distinct epoch in the study of the disease. An exhaustive bibliography is appended thereto.

NOTES FROM GERMANY ON THE VALUE OF RÖNTGEN RAYS IN MEDICAL DIAGNOSIS.

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DURING a year spent in Germany nothing impressed me more than the extensive employment of Röntgen rays in the examination of medical cases and the better results obtained with their aid than I had been accustomed to see in England. This is, no doubt, largely due to the fact that a number of the most distinguished German physicians have themselves paid much attention to the practical details of Röntgen-ray work, instead of leaving the examination of their patients, as is usual in England, to the X-ray specialist, who interests himself in surgical as well as medical patients, and sees little of the clinical and pathological aspects of the cases which he is called upon to investigate.

All patients are first examined by the fluorescent screen, as in thoracic and abdominal cases, this method gives information which cannot be obtained from a skiagram. A diagram is often subsequently taken, and in the hospitals a reduced positive is printed of a convenient size for fixing in the clinical report. In Munich and Berlin the negatives are exhibited at the clinical lectures, which take the place of the bedside teaching of English hospitals, by means of an ingenious arrangement by which they can be well seen by everybody in a large lecture room. On one side of a square wooden box containing an electric lamp there is a plain glass window, into the frame of which the plates fit; when the electric light is turned on and the room is darkened, details are often clearly visible, which can scarcely be made out in the positive or by the ordinary means of examining a negative.

A third method of examining cases, which has been of very great service, is by means of orthodiagraphy, a method invented by Professor Moritz[1] of Giessen. So far as I am aware, orthodiagraphy has not yet been employed to any extent in England; a short description of the method may therefore be of interest. With the ordinary X-ray apparatus the shadow formed is larger than the object and does not accurately reproduce its shape, because the rays which cast the shadow are divergent and not parallel. By means of orthodiagraphy, however, the exact size and shape of an organ or tumour can be ascertained. In its simplest form (Fig. 1) the apparatus consists of three metal bars fixed rigidly so as to form three

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[1] *Munch. med. Woch.*, 1900, p. 992, and 1902, p. 1; *Deutsches Arch. fur klin. Med.*, LXXXI., 1, 1904, and LXXXII., 1, 1905.

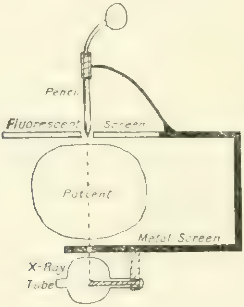

Fig. 1.

sides of a square. One of the parallel bars supports the X-ray tube and the other the fluorescent screen. The former also carries a metal screen which has a small hole in it situated over the anticathode of the tube. A pencil, which points directly towards the anticathode, is fastened behind the fluorescent screen, through a small hole in which it can be caused to project by a pneumatic arrangement. By this means all rays from the tube are cut off except a small bundle, which passes directly towards the pencil and traverses the body of the patient, who stands or lies between the parallel bars. The whole system of tube and metal screen, pencil and fluorescent screen can be readily moved in all directions in vertical planes if the patient is standing, or in horizontal planes if he is recumbent.

On moving the pencil along the border of the shadow of an organ such as the heart, and periodically causing it to project through the hole in the fluorescent screen so as to mark the patient's chest, the exact size and shape of the organ is mapped out, as, owing to the corresponding movement of the tube and metal screen, the rays in each position of the pencil are parallel with each other. With the best apparatus the outline can also be directly marked on a sheet of paper over the screen, or still better on a card fixed behind the patient by means of a second pencil, which can also be moved in the orthodiagraphic axis by a pneumatic arrangement.[1] By moving the apparatus so that the front pencil successively points towards the suprasternal notch, the nipples and various points on the chest outline and the costal margin, the position of these can be marked on the card by the second pencil, so that the relation of the heart to the chest wall can be well seen. By this means a permanent record of the result of the examination is obtained, and it is possible to overcome the difficulty of exact measurement on a curved surface such as the chest, a difficulty which increases as soon as the left margin of the heart extends beyond the nipple line. Moreover, the error introduced by the mobility of the skin, especially in women with pendulous breasts, is avoided.

I shall now describe some of the results which have been obtained by the various methods of examination.

Circulatory System.—The normal shadow of the heart and great vessels shows two convexities on the right side, formed by the vena cava superior above and by the right auricle below, and three on the left, the uppermost being formed by the arch and beginning of the descending part of the aorta, the lowest by the left ventricle and the middle small curve by the infundibulum and pulmonary artery, and occasionally by the left auricular appendix. The approximately accurate outline of the heart, given by an ordinary skiagram, is often useful as a permanent record, by means of which the relative development of dilatation and hypertrophy of the different parts of the organ can be traced as the disease progresses. The skiagram is especially valuable in the case of the middle curve of the left side, as it is impossible by any other means to define accurately how much of an area of dulness to the left of the sternum in the upper intercostal spaces is due to dilation of the infundibulum.

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[1] An apparatus of this kind has been devised by Dr. Groedel III. of Nauheim, and was exhibited by him at the Congress for internal medicine, held in April of last year in Munich.

Changes in the size of the heart, too slight to be appreciated by percussion, can be determined by the system, devised by Moritz, of measuring orthodiagrams which have been taken direct on paper. Fig. 2 is a reproduction of a tracing obtained by this method, and the three dotted lines show the most important measurements; *a* is the length of the heart, measured from the apex to the angle between the outline of the right auricle and that of the vena cava superior; *b* and *c* are respectively the greatest distances of the right and left borders of the heart from the middle line of the body. The normal average lengths in centimetres of *a*, *b* and *c* in adults are 13·5, 4·5 and 8·5 respectively. Somewhat considerable variations from the average may occur in normal people, depending mainly on the height and muscular development of the individual, but partly on conditions which are still undetermined. The measurements in women do not appreciably differ from those in men, except the average for *b*, which is 1 cm. less than in men.

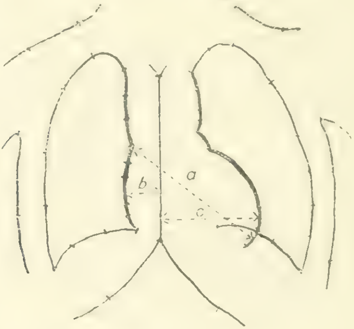

Fig. 2.

These measurements in one plane are of much greater value for purposes of comparison than those usually made along the curved surface of the chest of outlines obtained by percussion, as the latter are considerably influenced by variations in the shape of the thorax, though the actual size of the heart is quite independent of this.

By means of orthodiagraphic measurements, the temporary acute dilatation of a normal heart, described by Schott and others as occurring after wrestling and bicycling, has been shown to be non-existent, even when the exertion is great enough to produce extreme dyspnœa, cyanosis, and an acceleration of the pulse rate to over 150 to the minute. The disturbed cardiac action, caused by over-exertion in patients convalescent from fevers, or suffering from various anæmic and cachectic conditions, is also accompanied by much less dilatation than the ordinary physical examination might suggest. The false impression produced in such cases is due to the increased area of the visible cardiac impulse, the outermost part of which can often be seen external to the nipple line. This need not, however, indicate any dilatation, as the outer limit of the impulse is often some distance outside the outer border of the heart as determined by orthodiagraphy, especially in pigeon-breasted individuals, in whom the heart strikes against a laterally inclined part of the chest wall, the thickness of which determines to what extent the visible impulse is external to the cardiac apex.

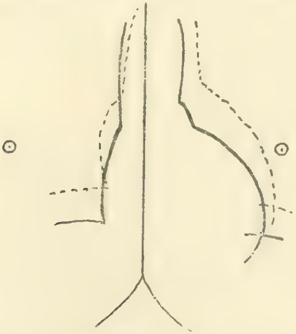

Fig. 3.

By similar means the influence of various forms of treatment on a dilated heart can be traced from day to day more accurately than by any other method. A comparison of the cardiac orthodiagrams of a patient, convalescent from an attack of endocarditis, myocarditis or heart failure, taken before and after slight exertion, shows whether the exertion is accompanied by any dilatation, and a guide to the amount of exercise which can be safely allowed is obtained.

Fig. 3 shows how very considerable the influence of posture on the shape and position of the heart may be, the continuous outline being the orthodiagram taken when the patient, who had a perfectly normal heart, was standing, and the dotted outline that taken when he was recumbent. In the vertical position the heart is always lower in the thorax and is generally narrower and longer than in the horizontal position. The area enclosed by the orthodiagraphic outline is diminished, and, as the thickness of the heart when examined from the side is either unaltered or diminished by the same change in position, the whole volume of the heart must be less when the body is in a vertical position than when it is horizontal. The effect of a vertical position is probably mainly due to gravity acting directly on the heart, and indirectly to the pull of the liver on the diaphragm and the attached pericardium, especially in individuals with lax abdominal muscles. The pull of the pericardium on the root of the great vessels lengthens and narrows them ; at the same time the tension of its lateral parts, which alone are unattached, produces a narrowing of the heart itself.

As the normal variations in the cardiac dimensions are much greater in the vertical than in the horizontal position, Moritz recommends that all orthodiagraphic examinations should be made in the latter position, and, for the same reason, percussion of the heart is best undertaken when the patient is lying down. Thus of two cases of dilatation, diagnosed in the horizontal position, one may remain obvious on standing, whilst, in the other, the heart may be apparently of normal size, owing to the greater narrowing it undergoes.

Orthodiagraphy also shows that deep inspiration gives rise to a well-marked movement of the apex of the heart downwards and inwards, and a smaller similar movement of the right cardio-diaphragmatic angle. These changes are, of course, mainly due to the alteration in shape of the thoracic cavity, caused by the diaphragmatic and costal movements, and only very slightly to the changes in the heart itself. In quiet respiration, the change in the position and shape of the heart is too small to interfere with the orthodiagraphic results, as the dome of the diaphragm does not alter its position relative to the chest wall, its contractions resulting merely in flattening of the muscular portion.

The difference in the cardiac outline in systole and diastole is much less than might be expected, the apex moving inwards during systole only half a centimetre. Owing to the diastolic pause, it is quite easy to mark out the diastolic position, which is invariably taken as the basis for orthodiagraphic measurements.

The relative accuracy of different methods of percussing the heart have been compared by means of orthodiagraphy. Of 120 cases, Moritz found that the left boundary of relative cardiac dulness, determined by moderately strong percussion, was within ½ cm. of that of the orthodiagraphic silhouette in 70 per cent., and the right boundary, determined by stronger percussion, in 68 per cent. Both boundaries were correct to this extent in the same individual in 50 per cent., and both were incorrect in 12 per cent. The error, however, was generally small, being greater than 1 cm. in only 9 per cent. on the right and 14 per cent. on the left side, but in one instance it was as great as 4·6 cm. and in another 3·3 cms., both being cases in which orthodiagraphy showed that percussion had exaggerated the size of the heart. Most of the cases, in which the error was greater than 1 cm., were patients who had spinal deformities, or emphysema, or were very fat. The discrepancy would be much more frequent if the results of percussion, obtained by those who maintain that the relative cardiac dulness does not normally extend to the right of the sternum, were compared with the outline obtained by orthodiagraphy, as the latter invariably reaches beyond the right sternal border.

A new method of percussion, which has, I believe, not yet been employed in England, has been recently advocated by Goldscheider,[1] and has been found to give a complete outline of the heart and great vessels, which invariably corresponds very closely to the orthodiagram. The percussion is so extremely light that it can be only just heard when the ear is placed near the chest. By this means, if it is begun outside the heart area, a just audible resonant note is obtained when vibrations are produced through the whole depth of the lung ; as soon as the edge of the heart, however deeply it may be situated, comes under the finger, the column of vibrating lung is diminished in length, so that the minimal resonant note disappears, and complete dulness results. Hence the outline is obtained as easily and as accurately in patients, with severe emphysema, as in those with normal lungs. The tip of the percussed finger alone touches the chest, the terminal phalanx being always held in a direction parallel to a line drawn perpendicularly to the sternum, so that in the lateral part of the chest it is not perpendicular to the surface. The terminal phalanx is flexed to as near a right angle as possible on the second phalanx, and the last interphalangeal joint is percussed. As the vibration in the chest produced by the percussion occurs mainly in lines, which have the same direction as the rays in orthodiagraphy, Goldscheider's method has been called ortho-percussion.

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[1] *Deutsche med. Wochenschrift*, XXXI., pp. 332 and 382. 1905.

The position of the percussed finger is particularly important when the heart is enlarged towards the left. For when the heart is percussed in the ordinary way—by strokes in a direction perpendicular to the chest—a relatively dull note can be obtained, owing to the nearness of the heart to the lateral chest wall, at a point which, viewed from in front, is quite outside the cardiac area. Hence, in such cases, the left border is always marked out too far to the left, an error avoided by sagittal percussion. The proximity of an enlarged heart to the lateral chest wall also explains why Moritz, using the ordinary method, found that the best results were obtained by moderately light percussion for the left border of the heart, though the right border required strong percussion.

The chief disadvantage of Goldscheider's ortho-percussion is that it requires an absolutely silent room, so that in an ordinary hospital ward or in the out-patient department it cannot, as a rule, be employed.

By the kindness of Dr. J. H. Pratt, of the Massachusetts

General Hospital, I was able to use the orthodiagraph in his clinic—the only apparatus of the kind in America—in order to test the value of auscultatory percussion in determining the size of the heart. The stethoscope was placed in any position on the chest wall, which was known to cover some part of the heart. The chest was then directly percussed along numerous lines from without inwards by a single finger, but always in a sagittal direction. The points, at which a distinct increase in the loudness of the note heard was observed, were marked, and the line, joining these together, was taken to represent the cardiac outline. In the somewhat limited number of cases which I examined, I found that the results obtained, both for the heart and great vessels, by this method were quite as accurate as by ortho-percussion, even in severe cases of emphysema.

In a case of left-sided pleural effusion, two independent observers found by ordinary percussion that the heart was so far displaced that the cardiac dulness appeared to extend to the right nipple line. By auscultatory percussion, however, I found that the cardiac border was situated two inches to the left of this line, a result which was subsequently confirmed by the orthodiagraph.

This method is easier to learn than ortho-percussion, and has the additional advantage that it can be carried out in the noisiest of rooms. Moreover, it affords the only method of ascertaining the position of the whole of the lower border of the heart. As the orthodiagraphic control shows that the right and left borders, marked out by auscultatory percussion, are correct, it may be presumed that the position of the lower border, found by exactly the same means and with equal facility, is also accurate, although it is impossible to prove this definitely, as the liver shadow generally prevents its determination by orthodiagraphy. Lastly, in cases of pleural effusion, in which the cardiac dulness and the dulness due to the fluid merge into each other, orthodiagraphy and ortho-percussion are of no more value than ordinary percussion in determining the position of the cardiac border, but auscultatory percussion gives a definite and probably accurate outline.

It is important to distinguish auscultatory percussion from auscultation of the sound produced by stroking the skin, a method which has been shown by de la Camp and by

Moritz, on comparison with orthodiagraphy, to be absolutely untrustworthy.

With regard to the great vessels, the orthodiagraph shows clearly when dilatation of the aorta is present. But skiagraphy is not as infallible an aid to the diagnosis of thoracic aneurysms as it might be expected to be. Professor Muller of Munich lays great stress on the misleading evidence afforded by visible pulsation. The aortic shadow shows much greater pulsation in cases of aortic incompetence than in aneurysm, and in two cases, which I saw, the complete absence of pulsation from the shadow led to the diagnosis of thoracic tumour, when an aneurysm was in reality present.

But, in many instances, the X-rays will clear up a doubtful diagnosis, or even occasionally reveal the existence of an aneurysm before any symptoms pointing to its presence have appeared. It is the practice in Munich to take skiagrams of doubtful cases in lateral and diagonal directions, as well as from in front and behind, as an abnormal shadow can sometimes be seen in one of the pictures which is absent from the rest. Moreover, a comparison of the different skiagrams makes it easier to ascertain whether the shadow is in continuity with that of the aorta or is separated from it, in which case the diagnosis of an aneurysm can be excluded.

Very valuable information can be given as to the progress of a case under treatment by periodically measuring the size of the shadow by orthodiagraphy. An increase in the opacity of the shadow is also important, as it indicates the occurrence of thrombosis.

It is well known that, in some patients, there may be considerable degeneration of the cerebral, cardiac, and renal arteries, although the superficial peripheral arteries are normal. In a number of cases, I have seen skiagrams of the legs showing well-marked calcification in the deeper arteries, such as the posterior tibial, although the palpable arteries were soft, and this has afforded useful evidence in support of the belief that some of the important internal vessels were in an abnormal condition.

Respiratory System.— Skiagraphy, as an aid in the diagnosis of phthisis, appears to be less frequently employed in Germany than it is in England. Unilateral limitation of the movement of the diaphragm probably due to latent pleurisy--can be watched on the fluorescent screen, and is recognised as sometimes pointing to the presence of tuberculous disease before any abnormal physical signs are present, but the excursion of the lower border of the lungs during deep respiration, as determined by percussion, probably yields equally reliable information. Diminished transparency of the apex of a lung only occurs when caseation is present, so that the early auscultatory signs probably always precede it. Moreover, a difference in the transparency of the two apices, just as a difference in the percussion note, may occur in normal individuals.

The value of the X-rays in diagnosing and watching the progress of cases of pleural effusion and pneumothorax is too well known to require much consideration here, though the importance of frequently repeated examination of the former with the aid of the screen, when, owing to thickening of the pleura and deposition of lymph, the physical signs give indefinite information as to the increase or diminution of the effusion, is often insufficiently realised.

The routine daily examination of a series of cases of pneumonia in Professor Muller's clinic in Munich by Professor Rieder has led to some interesting conclusions. In early cases, in which the symptoms point to pneumonia, although no signs of the disease can be found, examination by the Rontgen rays can always settle the diagnosis, as even consolidation, commencing at the root of the lung, leads to the formation of a definite shadow and often leads to deficient diaphragmatic movement. This is of particularly great value in cases in which it is doubtful whether abdominal symptoms are due to peritonitis or to deep-seated pneumonia. The shadow, which is always more extensive than the area of dulness, constantly increases in size until the crisis comes, thus differing from the area of dulness, which may remain constant, or, according to some observers, even diminish before the temperature begins to fall. This observation disposes of the claim, made by the advocates of various methods of treatment, that their specific stops the spread, or actually diminishes the area of consolidation without shortening the duration of the pyrexial period, as the daily increase in the area of the shadow, until the crisis comes, is an absolutely constant feature of the disease. The commencement of consolidation on the opposite side to that primarily affected can often be more early recognised by the aid of the rays than by other means, as a slight diminution in resonance cannot always be distinguished, owing to the absence of a normal side for comparison. Complete resolution of the affected lung is much slower than is generally believed, as there is always some diminution in transparency, and some impairment in the diaphragmatic movement for several days after the physical signs are completely normal. When dulness on percussion remains for some days after the crisis, it is often difficult to ascertain whether the cause is delayed resolution or accumulation of fluid in the pleural cavity. Examination with the screen can always settle the question, as a clear area between the diaphragm and the shadow in the lungs shows with certainty that no fluid is present.

The most remarkable observation made in the investigation of this series of cases was that of the development, in two instances, of an abscess in the pneumonic lung. In neither case were there any signs or symptoms pointing to the presence of the complication, with the exception of slight pyrexia and a very small quantity of purulent but non-fœtid sputum which contained no elastic fibres. Complete recovery took place in both cases after some weeks. The appearance on the screen, which led to the diagnosis of abscess being made, was very characteristic. An indefinite dark circle appeared in the area, where the darkness, produced by the pneumonic consolidation, was gradually clearing away. The interior of this circle was clearer than the surrounding area, but the

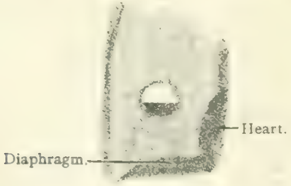

Fig. 4.

lower half was filled by a dark shadow with a horizontal upper border. (Fig. 4.) In the case, which I had the opportunity of examining myself, it was quite easy to see this border become irregular when the patient shook himself, and it was always the lower part of the circle which was filled, in whatever position the patient was placed. Clearly nothing but an abscess could give rise to this appearance. The fluid was observed to gradually diminish in quantity and the circle became smaller, until both finally disappeared. Now it is usually said that an abscess is a very rare and very fatal complication of pneumonia. But here were two cases, discovered in a comparatively small series, and in both the abscess gave rise to no signs or striking symptoms, and was completely absorbed. It is thus possible that abscesses, running a latent and favourable course, are a not infrequent complication of lobar pneumonia.

The evidence for the presence of bronchiectasis—often more or less uncertain—can be greatly strengthened by an examination with the X-rays, and I have seen several skiagrams which have been the means of confirming a doubtful diagnosis.

Tuberculous mediastinal glands are notoriously difficult to diagnose with certainty in a large proportion of cases, but they can be recognised, without much difficulty, by the X-rays, as ordinary inflamed glands form no shadow, whereas caseous glands give shadows whose position and shape are quite characteristic.

Digestive System.—The pioneer work of Cannon,[1] at Harvard University, in investigating the movements of the food through the normal alimentary canal by means of the X-rays, has recently been extended, with admirable results, by Rieder in Munich and Holzknecht in Vienna to the study of pathological conditions.

In cases of obstruction to the œsophagus, in which, for any reason, the use of a sound is considered inadvisable, the exact localisation and the degree of the obstruction can be easily ascertained by watching on the screen the passage of a bolus, and of a mouthful of fluid, containing bismuth subnitrate, which is very impervious to the rays, the patient being in such a position that the rays traverse the œsophagus from side to side unobstructed by the spine or aorta.

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[1] *Vide* an excellent summary of his work by Cannon in *The American Journal of the Medical Sciences*, April, 1906

In a remarkable case, which I saw in Professor Muller's clinic, a man could swallow liquids easily, but solids produced a painful sensation under the sternum, and were rejected in large quantities some time after eating. Food, mixed with a bismuth salt, could be seen to stop at a point near the cardia, the œsophagus above this being enormously dilated. An ordinary sound was stopped at the same point, but the probably functional nature of the case was shown by the ready passage, which could be watched with the screen, of a large india-rubber tube closed at one end and filled with mercury, the obstruction yielding instantaneously to the heavy and flexible sound formed by the tube with its contents.

In order to examine gastric cases, half a pint of bread and milk, containing about one ounce of bismuth subnitrate, is given on an empty stomach. This large quantity of the salt is never found to do any harm or to produce any discomfort ; but it is usual to add some lactose in order to prevent the possible occurrence of constipation. When the "bismuth meal" is finished, the patient is examined with the aid of the screen at regular intervals of half an hour or an hour, and, if necessary, skiagrams are also taken. If the X-ray tube is sufficiently good, the shadow of the stomach is very clearly seen, so that the size and shape can be ascertained with far greater precision than by any other means. When great accuracy is required, orthodiagraphy may be employed. Whilst the rest of the stomach is seen as a shadow, the fundus appears as a clear circular area, as, even when the stomach is full, there is a collection of gas in this situation.

Professor Rieder has found that, in no less than 80 per cent. of normal individuals in the erect posture, the stomach is vertical in position, the lesser curvature being situated almost exactly over the left border of the bodies of the vertebræ. Thus the whole stomach is situated in the left half of the body, except when the organ is full, in which condition, the pylorus is just to the left of the middle line.

As the lesser curvature is as distinctly visible as the greater curvature, it is easy to distinguish gastroptosis from dilated stomach, though percussion, which in both cases often only shows the low level of the greater curvature, may leave the observer in doubt as to which of the two conditions is present. An hour-glass stomach can also be more easily diagnosed by skiagraphy than by any other means.

The motor functions of the stomach can be accurately investigated by means of the screen, as the peristaltic waves can be seen passing down the curvatures of the organ. Cannon[1] has shown that the stomach consists of two parts with distinct functions. In the pyloric part, the food is broken up and thoroughly mixed with the gastric juice by peristaltic waves, three of which pass to the pylorus every minute; when the valve relaxes, the food is pressed into the duodenum by the same mechanism. No peristalsis, however, occurs in the cardiac part, which acts merely as reservoir for the food, in which salivary digestion can continue to be active, as the reaction in its centre may remain alkaline for two or more hours. As the pyloric part empties, increase in the tonic contraction of the cardiac part causes its contents to pass gradually into the region of active gastric digestion.

In cases of motor insufficiency of the stomach, the fluorescent screen gives a means of comparing the frequency and strength of the peristaltic waves with the normal, and diagnosis between atonic dilatation and dilatation from pyloric obstruction can also be easily made. The value of abdominal massage in atonic conditions can be demonstrated, as it is seen to be immediately followed by an increase in the vigour of the peristalic contractions.

Normally, all the bismuth disappears from the stomach in about three hours, with the exception of a very small residue, which can generally be seen in the most dependent part of the organ for several hours after the meal. By periodical examination, it is possible to ascertain to what extent stagnation of the gastric contents is present, as it has been proved, by comparison with the results obtained with the aid of the stomach-tube, that the bismuth has no influence on the motor functions of the stomach.

The small intestines cannot, as a rule, be very definitely seen, owing to the rapid passage of the bismuth through them, and the diminished concentration of the salt, due to the large quantities of digestive juices present. But in the rare cases of obstruction in the small intestine the salt becomes concentrated in the part above the obstruction, so that its position can be easily defined.

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[1] *American Journal of Physiology*, I., p. 367, 1898

The large intestines can be examined by the same "bismuth meal" as the stomach, as the slower passage and greater concentration of the contents cause the large intestine to be almost as clearly marked out as the stomach. But, owing to the two or three hours required for the bismuth to reach the cœcum, it is more convenient, as a rule, to examine the colon by giving a rectal injection, consisting of a pint and a half of milk or water, containing three ounces of bismuth subnitrate in suspension. It is best previously to empty the large intestine as thoroughly as possible by means of an ordinary enema.

The X-rays show that a rectal tube cannot be passed higher than the lower part of the sigmoid flexure, as it becomes doubled on itself, when an attempt is made to reach the ascending colon. However, an injection of a pint and a half of a bismuth suspension through a tube in the rectum always renders the cœcum visible, but never the ileum or appendix, a fact which confirms the general belief that medicinal enemata, if sufficiently large, may be of value for the whole colon. Cannon has further shown, by observations on animals by means of the Röntgen rays, that a large injection of the consistency of thick cream gives rise to anti-peristaltic waves in the ascending colon, which may actually cause all the enema to enter the ileum. Under normal conditions, however, the antiperistalsis, which Cannon has shown to be constantly present in the ascending colon, does not press any of its contents through the ileo-cœcal valve, but merely serves to mix it thoroughly with the digestive secretions brought from the small intestine, and to expose it to the absorbing mucous membrane of the ascending colon.

It is easy to determine the exact position of a chronic or partial obstruction of the colon after either a bismuth meal or a bismuth enema, and, in doubtful cases, obstruction can be distinguished from severe constipation due to other causes. Fig. 5 is a sketch of the skiagram obtained after a bismuth meal in a case of chronic obstruction ; it is seen that the obstruction, which is incomplete, is in the ascending colon, and that the remaining portion of the ascending colon, as well as the hepatic flexure and the transverse colon, are collapsed.

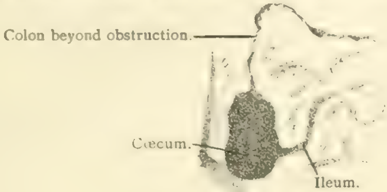

Fig. 5.

The operation, subsequently undertaken, showed that the obstruction was due to a cicatrised tuberculous ulcer, the colon, beyond the obstruction, having contracted to the thickness of a finger.

The exact position of the transverse colon, in cases of enteroptosis, is readily made out by similar means. Palpation of the abdomen, during examination with the screen after a bismuth meal, gives valuable evidence as to the localisation of tumours, as from the position of the hand palpating the tumour it can be seen whether the latter is within the area of the stomach or colon shadow.

In the application of the Röntgen rays to the diagnosis of renal calculi and diseases of bones and joints, I do not think that we in England have anything to learn from Germany. But I hope that I have succeeded in this paper in showing how, in the Rontgen rays, we have a means of extending our knowledge of diseases of the thorax and abdomen, which until now has not in England received the attention it deserves.

THE USE OF ANTIPYRIN AS A SEDATIVE IN SOME OF THE COMMON DISORDERS OF INFANCY AND CHILDHOOD.

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MUCH has been said and written of the ill-effects which may follow the administration of members of that series of drugs, which includes phenacetin, antipyrin, antifebrin, and the like, and especially of their depressing action on the heart.

Originally introduced as antipyretics, their employment as such has largely, and very rightly, fallen into disrepute.

As analgesics they are undoubtedly dangerous, unless used with due caution. The frequency with which they are prescribed for the headache and neuralgia of women, the demand for repeated doses for these constantly recurring and distressing ailments, the all-too-common tendency for patients to take such drugs without medical advice, and without regard to dosage, these and other causes have resulted in a considerable number of instances in which grave ill-effects have followed their use. Consequently, a prejudice has arisen against them, they are regarded by many as being dangerous, and are not employed in some conditions in which they might be of the greatest service.

It is the object of the present writer to record the beneficial results, which have been observed in the employment of one of them, *antipyrin*, as a *sedative* in some of the nervous disturbances of childhood. This special action of the drug is, of course, no new thing, and has been recommended by high authorities, but somehow or another, possibly on account of the reasons before instanced, it has not met with the general recognition which its great value certainly justifies.

During an epidemic of whooping-cough in the spring of this year, we had many cases brought to the Children's outpatient department of St. Mary's Hospital, Manchester. It is beyond the scope of this paper to discuss the ætiology of whooping-cough. The specific bacillus is said to have been recently isolated from the tough mucus which is expectorated in the early days of the disease, but this still requires confirmation. Whatever may be the nature of the illness, whether it is a toxæmia, or a lesion located in some part of the respiratory tract, which reflexly causes cough and vomiting, the harmful effects, at any rate, appear to be secondary to two factors, (1) the *cough*, causing, as it does, exhaustion, loss of sleep, and also grave structural changes in the lungs and chest wall, and (2) the *vomiting*, which interferes with nutrition, and may even cause inanition. It follows, therefore, that, in the absence of any pathological basis of treatment, our energies must be chiefly directed towards the alleviation of these two symptoms.

In this epidemic, various sedative drugs, such as belladonna, potassium bromide, chloral, antipyrin, etc., were tried in conjunction with an expectorant mixture. The usual experience was met with, viz., that one drug would often influence a case in which the others almost entirely failed. But of all the drug tried, undoubtedly the most serviceable was antipyrin.

In no case could it be said to have cut short the disease, but it usually influenced the two symptoms before mentioned, viz., the paroxysms of coughing and the vomiting following them, in a marked manner. For the latter, in the present epidemic, at any rate, it was practically a specific. It seldom failed, after two or three doses, to stop the vomiting, and that even in cases in which it did not appear to influence the paroxysms themselves. In most instances, however, the paroxysms were considerably modified. After several doses of 1 or 2 grains, given every 4–6 hours, the frequency would diminish to half or even less. By doubling the dose at bedtime, a comparatively quiet night's rest was often obtained. As a result it was, in many cases, easier to maintain the nutrition of the little patient, his strength was conserved, sleep was less interrupted, and we may reasonably infer that the damage to the lung tissue was slighter. The dose, which we have usually found efficient, has been from 1 to 3 grains, four to six times daily, according to the age of the child and the severity of the attack.

The beneficial action of antipyrin, as a sedative, is well exemplified in its effect on the nervous irritability which frequently accompanies teething. Although many of the ailments, which occur during the first dentition, and which are frequently ascribed to it, are in reality due to entirely different causes, such as improper feeding, rickets, etc., yet there remains a proportion of cases, characterised by nervous irritability, pain, sleeplessness, and the like, which, if not actually due to the cutting of the teeth, are at least intimately associated with it. And it is here that antipyrin, given in doses of 1 to 2 grains two or three times daily, produces such good results. The occurrence of a certain number of cases, in which the eruption of each new tooth, or set of teeth, is accompanied definitely and regularly by vomiting, diarrhœa or cough, and occasionally eczema, suggests that teething may be the predisposing cause. Its action may be reflex, or may determine, for the time, such nervous instability that slight irritation in the respiratory tract, or in the alimentary canal, will produce effects quite out of proportion to its magnitude. In several such cases, the symptoms have been checked by the employment of antipyrin alone. In others, it must be confessed that it has had little effect, probably owing to the fact that the cause lay elsewhere. This heightened irritability of dentition occurs especially in children of a nervous diathesis. It is also characteristic of rickets, and, although the nervous symptoms, occurring during the dentition of a rachitic child, should first of all be treated on antirachitic lines, yet they will be more rapidly and effectively alleviated by small doses of antipyrin in addition.

In three cases of night terrors, in which we used antipyrin, the results were so striking that they may be worthy of record. In one, we were called to see a boy of 11, who was just getting over a severe attack of tetany, the fourth during his life. He was a highly strung, nervous lad, with a history of fits during infancy. For a year past, he had had terrors practically every night, and often, though not always, enuresis. No predisposing cause, such as adenoids, worms, etc., was discovered. His bowels were opened, and he was given 2 grains of antipyrin, three times daily, the last dose just before going to bed. On the very first night after taking the powders, he was without the terrors and slept peace-fully. He continued with the powders for a fortnight, and measures were taken to improve his general health. On seeing his mother, three months afterwards, she informed us that he had had no further terrors, or enuresis, after the first day of taking the antipyrin.

In this case, due credit must of course be given to the cod-liver oil and malt, fresh air, and absence from school, etc., which much improved his health, but the sedative effect of the antipyrin was, undoubtedly, an immediate and important factor in this satisfactory result.

The second case was that of a lad of 8, also with a history of convulsions in his first two years of life, and, as his mother remarked, "terrible nervous." For three months previous to coming to hospital, he had had enuresis every night. He would suddenly sit up in bed and scream as if in great terror, pass his water under him, and then spring out of bed, and give vent to piercing shrieks. He was put upon antipyrin alone, in 2-grain doses, three times a day for a fortnight. On the second night, the enuresis and terrors stopped and he slept quietly. During the six weeks, which have now passed since treatment was commenced, the enuresis has been entirely absent, and the night terrors have recurred twice only. In this case, all other treatment, save that by antipyrin and exemption from school, was withheld for the first fourteen days, in order to give the drug a fair trial, and, in this fortnight, he had a peaceful sleep every night.

The third case was somewhat different. A child, aged 2½ years, was brought up because she had never progressed satisfactorily with her walking. It was found, on examination, that she was suffering from a spastic condition of both arms and legs, not marked except in the left leg. On enquiry, it was found that her birth had taken place after a lingering labour, and that she had always been a very nervous child. She had had restless sleep, often broken by her sitting up suddenly while asleep, and screaming. She was often dull and heavy in the morning, and seldom seemed refreshed by her sleep. Her face and behaviour gave one the impression that cerebral development was not satisfactory. She was given bromide of potassium for a fortnight with no result. She was then given antipyrin in 2-grain doses, three times daily, for the next fortnight. After a few days, there were no further terrors, and the child slept quietly. Her mother seemed much pleased and volunteered the information that she had been quite markedly brighter, and had waked up in the morning much more refreshed than before, and stated further that her appetite had noticeably improved. This latter was probably brought about by the quiet nights. bringing, as they would in their train, an improvement in general health.

There can be no question as to the vital importance of quiet undisturbed sleep, if the state of health both of body and mind is to be kept up to the proper pitch, and to no one is it more essential than to the growing child, particularly if he or she is not strong otherwise.

The help that may be rendered temporarily by a sedative, such as antipyrin, is well illustrated by the foregoing examples. Such sedatives, however, should be used with discretion, and never continued for a long time together.

The true cause must always be carefully sought for and treated, whether it arises, as in the first two of these cases, from a nervous temperament and depressed health, or from some derangement of the digestive apparatus, rickets, congenital syphilis, adenoids, or from some other of the many conditions, which cause or contribute to the disturbance of sleep.

Enough has been said to illustrate the great value and powerful action of antipyrin as a sedative in childhood. Its precise mode of action, and the part of the nervous system, which it influences, are not known. It has not the dulling effect on the cerebral cells which, for instance, morphia possesses. It does not appear to affect cerebral or motor activity to any extent, but rather to have some selective action in controlling and rendering less active sensory stimuli. Although it is regarded by many as a dangerous drug, the present writer has not observed a single instance, among the several hundred cases in which he has employed it, in which any ill-effects have followed its administration, and in no case has a rash resulted.

It is free from the hæmolytic action which all other members of this group unfortunately possess. The cyanosis, for instance, which may follow excessive use of phenacetin, or antifebrin, due to hæmolysis and formation of methæmoglobin, does not result from the administration of antipyrin. Again, it has no injurious effect upon the metabolism of healthy tissues as has antifebrin. If given in small doses, *e.g.*, one to two grains, its employment is almost entirely free from danger, and it only needs to be used in such cases, as have here been cited, to convince one of its efficacy, and to cause one to look upon it as an invaluable ally in the treatment of the nervous disturbances of childhood.

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DISEASES OF THE STOMACH.

A REVIEW OF SOME RECENT WORK.

(*Concluded.*)

By F. CRAVEN MOORE, M.Sc., M.D., M.R.C.P.,
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GASTRIC ULCER.

A CONSIDERABLE number of papers, dealing with various aspects of this recondite lesion, have appeared in the literature, but, with the exception perhaps of its treatment, without adding much to our knowledge.

Hæmorrhagic erosions of the gastric mucosa have been regarded as possible starting points for the origin of the round ulcer of the stomach, and quite recently Smith and Miller,[25] at the British Medical Association, expressed the view that the essential point in the first stage, in probably all cases of gastric ulcer, is inflammation of the lymphoid follicles in the mucous coat, and that subsequent events, hæmorrhage, gross ulcer, perforation, etc., depend entirely upon the destructive power of the inflammatory factors versus the reparative powers of the gastric mucosa. Funke,[26] on the other hand, maintains that erosions and gastric ulcer have no real relation. Conditions, having much more analogy to gastric erosions, are erosive gastritis, ulcerative stomatitis and ulcerative (but not follicular) colitis. Thrombosis and embolism are but rarely found, erosions are generally a consequence of an existing secondary gastritis, occurring in the course of various diseases, and induced by the excretion into the stomach of hurtful substances.

Hurwitz[27] looks upon hæmorrhagic erosions as an independent affection of the stomach, suddenly affecting the whole, or, at least, areas of the gastric mucosa and proceeding to necrosis. Digestion of the necrotic foci, opening up the adjacent capillaries, leads to hæmorrhage and to the clinically characteristic "coffee-ground" vomit. Microscopic investigations have failed to reveal any definite ætiological factor; certain it is that thrombosis is absent in the majority of cases. The writer is disposed to the view that the condition depends on irritation of the abdominal sympathetic, which produces spasm of the gastric vessels, and the areas of gastric mucosa, so rendered anæmic, are digested by the gastric juice.

It has long been recognised that sudden and severe hæmorrhage from the mucous membrane of the stomach may occur, especially in young females, without obvious cause, and in the absence of any gross lesion discoverable on inspection. Hale White[28] has collected, from his own experience and from the literature, 29 such cases, in all of which the absence of gross lesion of the gastric mucosa was verified by inspection, either during operation or post-mortem. On the basis of these, he concludes that there is a disease, which he names *Gastrostaxis*, occurring principally in young females, of which the most prominent symptom is hæmatemesis due, not to ulcer, cirrhosis, malignant disease, heart disease, or any commonly recognised cause of hæmatemesis, but probably to oozing of blood from the gastric mucosa; and that many cases, which the older writers and even Kuttner, so recently as 1897, described as vicarious menstruation, were examples of this disorder. Of his 29 cases, 27 were females and 2 males, the ages ranged from 18 to 54 years, the youngest being a male; 12 of the cases were included between the ages of 21 and 28 years, and 11 more between the ages of 30 and 39 years.

Clinically, gastrostaxis is characterised by the occurrence of attacks of hæmatemesis, extending over years with intervals of months, or a year or two between. Each attack consists of a series of occasions, separated by a few hours or a few days, when the patient brings up blood. The amount of blood vomited may be slight, or so great as to call for surgical interference, as happened in seven of the cases.

The bleeding may be manifest as melæna, and, in one case, in which the blood oozed from the duodenal as well as from the gastric mucosa, it was of a bright red colour as it was passed by the bowel. In addition, the patients may suffer from pain in the stomach after food, tenderness, and vomiting without blood. These dyspeptic manifestations may recur at intervals over a period of years, they may precede hæmatemesis, or this latter may be the first symptom. The patients generally appear better nourished than do cases of gastric ulcer, they are often anæmic and may have amenorrhœa. As to the cause of this disorder, Hale White's suggestions are entirely negative. He is convinced that it is not vicarious menstruation, that it does not depend on chlorosis that it is not neurotic, that it cannot be attributed to hæmorrhagic erosions. With regard to the latter, his evidence is not so convincing, he points out the much higher age incidence of the scattered hæmorrhagic erosions which were met with in 0·7 per cent. of 2,246 autopsies at Guy's, and their association with heart failure, abdominal disease, pneumonia, tuberculosis, arterial degeneration; but, from the observations of Dieulafoy, Mayo Robson, Dawson, Herringham, Stevens, and Miller (*vide supra*), it would appear probable that minute erosions of quite another type may occur, and be productive of the disorder described as gastrostaxis. As regards frequency, Hale White believes the condition to be much more common than is generally recognised, that, in fact, many cases of hæmatemesis in young females, usually regarded as significant of gastric ulcer, really belong to this category, and that it is the confusion of this disorder with gastric ulcer that is responsible for the usually accepted belief that gastric ulcer is more common, and occurs at an earlier age in females than in males. In support of this, he points out that, even in such statistics as show a greater mortality from gastric ulcer in females than males, this excess is never so great as it ought to be, if all cases of haematemesis in females were due to gastric ulcer, especially as post-mortem records show that hæmorrhage from an ulcer is a rare cause of death in young females. The differentiation of gastrostaxis from gastric ulcer he regards at present as almost impossible. The condition is but rarely fatal, and is to be treated by rest in bed, the administration of Liquor ferri perchlor., 30 with an equal amount of glycerine, to be repeated, if it does not cause sickness, or 5 to 10ɱ of 1 1,000 adrenalin solution, or calcium chloride grs. 10, three times a day. Rectal feeding should be continued for from two to four days after bleeding has ceased, when the patient may take a milk diet.

The ætiological significance of trauma and continuous pressure in the origin of gastric ulcer is dealt with by Ackermann.[29] From a large series of cases he has collected 16, which appear to have been so caused, 15 by compression, 1 by trauma.

Fertig[30] records the following example of traumatic ulcer:— A stableman received a kick on the epigastrium, his stomach being full at the time. Laparotomy was performed, but the external aspect of the stomach and intestine was found to be normal. Five days later, there was hæmatemesis and melæna, and death ensued the following night. The autopsy showed the existence of four ulcers on the smaller curvature.

From a microscopical study of gastric ulcers in the calf's stomach, Bloch[31] concludes that bacteria play no part in their origin or development, that interference with the circulation, in localised areas of the mucosa, is in itself insufficient to lead to the formation of a gastric ulcer, and that the essential starting point of this lesion is a loss of surface epithelium. This mucus-clad layer of epithelium protects the underlying tissues from the eroding action of the gastric juice, and a defect in it, at any place, allows of the deeper structures being eroded, and the formation of a typical round ulcer. The peculiar tendency to the localisation of ulcers in the vicinity of the lesser curvature, he explains by certain anatomical peculiarities. The less curvature is relatively firmly fixed, in the normal contractions of the stomach it has but little range of movement, the mucosa in this region is less mobile and smoother than on the greater curvature. Accordingly any deficiency of the surface layer of epithelium cannot be compensated, as it is in the region of the greater curvature, by contraction of the muscular coats and puckering of the mucosa.

The bacterial origin of the gastric ulcer has been recently investigated by Turck,[32] and he records typical examples of this lesion as occurring in the stomach of the dog following the administration of emulsions of the Bacillus coli by the mouth.

MacNevin and Herrick[33] give the following interesting analysis of 100 fatal cases of gastric ulcer:—

The location of the lesion, as noted in 97, was, lesser curve 47, posterior wall 30, anterior wall 17, greater curve 3.

Dividing the stomach, according to Mayo, by a vertical line to median side of the cardia into a pyloric and a cardiac portion, the ulcer was situated in the former in 94·3 per cent. and in the latter in 5·7 per cent. Symmetrical ulcers or scars were noted in 37 per cent., from which they conclude that the so-called recurrent ulceration is due to the formation of a symmetrical ulcer rather than to the recrudescence of an old scar, and this must be allowed for in statistical evidence of the results of medical treatment. The cause of death was noted in 55, as hæmorrhage in 25, 19 males and 6 females, all over 30 years of age except 1 female; and peritonitis in 30, 8 males and 22 females, 2 males and 13 females being under 30, 6 males and 9 females over 30 years of age.

In view of the significance which Mayo Robson attaches to a localised deposit of fibrin on the serous coat of the stomach as diagnostic of ulcer, it is interesting to note that they found such to be present over the base of the ulcer without perforation or adhesions in 74·3 per cent.

On the basis of 28 cases operated on by him, Riedel[34] gives the following as characteristic features of ulcer situated in the middle portion of the stomach. They show a greater tendency to the formation of a palpable tumour, than ulcers about the pylorus, and specially so, in such as are situated on the smaller curvature, and the inferior aspect of the anterior wall to the left of the middle line. Prior to the development of a palpable tumour, such ulcers are characterised by pain in the left side under the costal margin, and occasionally, by pain in the back about the eleventh rib. At first the pain is intermittent, later becoming more frequent and then continuous. Vomiting is frequent and a daily feature, but hæmatemesis occurred in less than half the cases. Finally, with healing, there results stenosis, and the production of an hour-glass deformity.

The origin of the pain in gastric ulcer has received various explanations. Formerly, on the supposition that the afferent nerves of the stomach were capable of conveying painful impressions, it was believed that the epigastric pain originated in the direct irritation of exposed nerves in the base of the ulcer (splanchnic pain of Ross), the dorsal pain (somatic pain of Ross) being referred. When, however, the fallacy of this supposition was demonstrated other explanations were advanced.

Mansell Moullin[35] regards the pain of gastric ulcer as depending on an inflammation of the lymphatics in the vicinity of the ulcer, the extension of which to the parietal peritoneum explains the tenderness on pressure.

According to Lennander[36] it is due either to adhesions, or to subserous lymphangitis about the cœliac axis, which renders the nerves of the abdominal wall so susceptible that pressure on their anterior and posterior branches is painful.

Mackenzie,[37] on the other hand, regards both the epigastric and dorsal pain, the cutaneous and muscular hyperæsthesia, as the expression of a viscero-sensory reflex. Abnormally powerful stimuli passing along the afferent nerves of the stomach to the spinal cord, initiated by irritation of their exposed terminal branches in the base of the ulcer, spread to, and affect adjacent spinal centres and so stimulating sensory, motor, and other nerves arising in this part of the cord, result in the production of pain referred to the peripheral distribution of the nerve whose spinal centre is stimulated.

As regards the relation between the location of the epigastric pain and tenderness and that of the ulcer in the stomach, Mackenzie has shown that pain in the upper part of the epigastrium occurs with ulcers of the cardiac portion of the stomach, in the lower part of the epigastrium with pyloric ulcers.

For the diagnosis of gastric ulcer, Hemmeter[38] proposes to utilise the X-rays in the following manner:—The patient takes on an empty stomach, before breakfast, a glass of warm water, in which is suspended a large teaspoonful of bismuth subnitrate, then, after reclining for half an hour either on the back, face, or left or right lateral position, according to the situation of the ulcer, the region of the stomach is examined by the fluorescent screen, when, if an ulcer is present, it is noted that in some part of the shadow of the stomach produced by the thin layer of bismuth subnitrate distributed over its mucosa, there is a dark spot produced by the accumulation of the salt in the cavity of the ulcer. Hemmeter states further that, whilst the general shadow of the stomach disappears with the passage of the bismuth into the duodenum, in from 3 to 6 hours, that produced by the bismuth in the ulcer persists for 24 to 36 hours. Should one examination fail to reveal the presence of an ulcer, the procedure must be repeated, with assumption of a different posture, after drinking the suspension, but not on the following day, as by that time the bismuth has only arrived in the transverse colon, and the shadow it produces in this position confuses the appearance.

As regards the treatment of gastric ulcer, the more notable innovations concern the dietetic *régime*.

In 1901, at the Congress for Internal Medicine, Lenhartz suggested a concentrated egg-albumin diet for the treatment of gastric ulcer, as one more suited to combat the hyperchlorhydria, and reinforce the enfeebled and anæmic state of the patient. The idea was put into practice in hospital and gave very good results. Details of the method, as given by Habermann,[39] are as follows :—Absolute rest in bed for at least four weeks. All mental excitement is to be avoided. An icebag is placed upon the stomach, and kept there almost continuously for two weeks, this prevents flatulent distension, promotes contraction of the stomach and alleviates pain. On the *first day*, even when hæmatemesis has occurred, the patient receives from 7 to 10 ounces of iced milk given in spoonfuls, and from 2 to 4 beaten eggs within the first 24 hours.

At the same time, bismuth subnitrate is given three times a day in doses of 30 grains, and is continued for 10 days. The eggs are beaten up whole (with a little sugar), and the vessel, containing them, is placed in a dish containing ice. A little wine may also be added. The amount of milk taken each day is increased by about 3 ounces, and the number of eggs by one, so that at the end of the first week, the patient is receiving up to 30 ounces of milk, and from six to eight eggs per day.

These are continued in the same daily amounts for another week. Some raw minced meat is allowed usually on the sixth day, about 1 ounce being administered in small quantities at a time. On the following day, the amount is doubled, and later may be still further increased if well digested. On the seventh day, some well-cooked rice and a few softened Zwieback are allowed. In the third week, a mixed diet is resumed. The bowels are not moved during the first week, and in many cases not until the end of the second week, small glycerine injections, or warm water enemata being employed. After the third week, this procedure is carried out daily if spontaneous

TABULATION OF THE LENHARTZ DIET FOR GASTRIC ULCER.

		Days after last Hæmatemesis.													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs*		2	3	4	5	6	7	8	8	8	8	8	8	8	8
Sugar with eggs	grammes	0	0	20	20	30	30	40	40	50	50	50	50	50	50
Milk	c.c.	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Raw chopped meat	grammes	0	0	0	0	0	35	70	70	70	70	70	70	70	70
Milk rice	"	0	0	0	0	0	0	100	100	200	200	300	300	300	300
Zwieback	"	0	0	0	0	0	0	0	20 = 1 piece.	2 pieces	2	3	3	4	5
Raw ham	"	0	0	0	0	0	0	0	0	0	50	50	50	50	50
Butter	"	0	0	0	0	0	0	0	0	0	20	40	40	40	40
Calories		280	420	637	777	956	1135	1588	1721	2138	2478	2941	2941	3007	3073

* From the first to the seventh day inclusively the eggs are beaten; from the seventh to the fourteenth day inclusively half are beaten and half are cooked.

evacuation does not occur. The patient is allowed to get up on the twenty-eighth day, and is discharged from the sixth to the tenth week.

Among others, Senator[40] has adopted the *régime* of Lenhartz, with certain modifications. He recommends a diet consisting of gelatine, fat, and sugar. Gelatine is easily digested, it possesses a high nutritive value, being a saver of proteid, it increases the coagulability of the blood, and thereby tends to still bleeding. Fat and sugar both possess high nutritive values, the former, in addition, exerts an inhibitory effect on the secretion of hydrochloric acid and a sedative influence on the base of the ulcer.

Cases admitted to hospital with recent hæmatemesis are placed at once on teaspoon doses of a decoction of white gelatine with sugar (gelatine 15 to 20 in 200 to 250 water and 50 Elæosacc. citri) administered every one to two hours, or, in severe cases, every quarter or half hour. Fresh butter and cream are also given in small frequently repeated quantities, so that in 24 hours the patient receives one ounce of butter and nine ounces of cream. If butter cannot be taken in its ordinary form, Senator gives it in small fragments, which are frozen hard, and which the patient sucks. The cream, which may, if preferred, be mixed with some sugar, can be whipped to a snow and kept on ice. In this way the patient, immediately following a hæmatemesis, receives food having a daily value of from 900 to 1,000 calories, double the number supplied in egg and milk diet of Lenhartz. If there is no recurrence of the hæmatemesis, milk, beaten eggs, and scraped raw meat are added to the dietary, the decoction of gelatine being gradually cut off, but at once resumed in the event of any subsequent hæmorrhage.

Ewald,[41] however, is of opinion that the giving of food by the mouth, however bland, immediately after a hæmorrhage, involves risks from distension of the stomach and displacement of the thrombus, which outweigh the advantages claimed for the Lenhartz method. In such cases, he withholds all food by the mouth for two or three days, and resumes oral feeding on the fourth day, beginning with milk and butter; on the ninth day he adds eggs, and, from the eleventh day onwards, he adds biscuits and gruel.

DUODENAL ULCER.

"The Duodenal Ulcer, by one of its Victims," is the title of a paper by Cooke,[42] in which he seeks to explain the pathogenesis of this lesion by the lack of antiferments in the cells of the duodenal mucosa, by which they are normally protected from self-digestion. "It is conceivable that in external burns, toxic substances are absorbed and are specific and destructive to the antibody in the epithelial cells of the duodenum. As soon as the antibodies are diminished to a sufficient quantity, an ulcer is the result." He offers, however, no explanation of the peculiar focal character of the lesion. Beyond this, his communication provides a useful summary as to the incidence of this lesion. The statistics of Moynihan and of Mayo show that the incidence of duodenal ulcer, compared with that of gastric ulcer, is much greater than is usually believed, and further, that the two lesions are not infrequently associated together (in 22 of 51 cases, Moynihan). The ulcer is situated in the first part of the duodenum ten times more frequently than in the second. In Collin's 262 cases, perforation occurred within two inches of the pylorus in 242; in the second part, in 14; and in the third part, in 6. Perry and Shaw found that out of 141 cases perforation occurred, in the first part, in 123; in the second, in 16; and in the third, in 2. In Moynihan's 51 cases, the ulcer was located, in the first part, in 50 (with seven perforations); in the second part, in 1 only.

In one of Moynihan's cases, two ulcers were present.

Most frequently, the ulcer is located on the upper portion of the anterior wall, thus explaining the frequency of general peritonitis in perforative cases. As regards sex, the relative preponderance of males over females is variously estimated at 1·5 to 1 to 6 to 1. The lesion has been recorded in an infant three days old (Lister and Spiegelberg). Collin, in 297 cases, found the age incidence to be as follows:—

Under 10 years - - - -	42 cases.
Between 10 and 20 years - - -	24 „
„ 20 „ 30 „ - - -	43 „
„ 30 „ 40 „ - - -	52 „
„ 40 „ 50 „ - - -	46 „
„ 50 „ 60 „ - - -	41 „
„ 60 „ 80 „ - - -	28 „
„ 80 „ 95 „ - - -	3 „

Of other ætiological factors, arteriosclerosis was found in 48·8 per cent. (markedly in 22 per cent.) of 82 consecutive cases of gastric and duodenal ulcers occurring at the Johns Hopkins Hospital (Howard). Hyperchlorhydria was present in only 26·4 per cent. of this series. And, in relation to this, Cook writes "any disproportion between the composition of the blood and gastric juice may give rise to ulcer, either in the stomach or duodenum by diminishing the antibodies in the epithelial cells. This disproportion may be due either to anomalies in the composition of the blood—anæmia, or to an increased acidity of the gastric juice. This explains the greater frequency of gastric ulcers in women, and of duodenal ulcers in men. It also accounts for the fact that gastric ulcer is usually not associated with hyperchlorhydria, being more common in women, the less frequent cases in men having this anomaly of secretion.

In a paper recording the details of 14 consecutive cases of perforated duodenal ulcer, Maynard Smith[13] gives the results of some interesting experiments he performed on the human cadaver, demonstrating the course taken by fluid escaping from a duodenal perforation in its most frequent situation (*vide* Collin). In every case he found that the fluid ran downwards in the direction of the right kidney pouch, and collected in a space delimited in *front* by the inferior aspect of the right lobe of the liver and the hepatic flexure of the colon; *behind*, by the anterior peritoneal-covered surface of the right kidney and the posterior abdominal wall; to the *outside* by the abdominal wall, and to the *inside* by the duodenum and the foramen of Winslow. *Upwards*, this pouch extends posteriorly between the right lobe of the liver and the diaphragm, as far as the inferior layer of the right coronary ligament, and more anteriorly and to the right of the coronary ligament, it extends between the upper surface of the right lobe of the liver and the diaphragm, as far as the falciform ligament. *Downwards*, the pouch is delimited by the reflection of the peritoneum from the hepatic flexure of the colon on to the anterior surface of the kidney and the second part of the duodenum. When this space had been filled, the further course of the fluid was always downwards along the outer side of the ascending colon as far as the brim of the pelvis, and, in this situation, it again accumulated until its level exceeded that of the pelvic brim, when it overflowed into the pelvis. In cases, in which the ascending colon was so bound down into the loin that its level was below that of the pelvic brim, the overflow occurred across the anterior aspect of the ascending colon, usually a few inches below the hepatic flexure, and thence, guided by the obliquity of the mesentery and the slope of the abdominal wall, it proceeded downwards and to the right to the appendix region, and, crossing the termination of the ileum, reached the right side of the pelvis practically in the same spot as before. The initial direction of the fluid, as it leaves the perforation towards the right, depends on the existence of a peritoneal reflection from the upper layer of the transverse mesocolon to the first part of the duodenum and stomach, which prevents it passing to the left. The fluid does not pass through the foramen of Winslow, because this opening is, both in the erect and recumbent posture, above the level of the fluid.

These observations were confirmed by the distribution of the suppurative peritonitis in cases of perforated duodenal ulcer, and they afford an explanation of such cases as simulate appendicitis, as happened in 19 of Moynihan's 51 cases.

NEOPLASMS OF THE STOMACH.

Cignozzi[41] describes an interesting example of *endothelioma* of the pylorus. The patient had suffered for 15 years from dyspepsia, and gradually developed signs of pyloric stenosis. Vomiting had occurred during the last 12 years but there was never hæmatemesis, or other signs of ulcer. The general condition of the patient gradually deteriorated, *pari passu*, with the defective ingestion and assimilation of nourishment, but cachexia was absent to the end. The pylorus presented a palpable tumour, smooth and movable. The stomach contents contained both free hydrochloric acid and lactic acid. At the autopsy, the pylorus was the seat of an annular stenosis, the adjacent portion ot the stomach wall, especially along the greater curvature and posteriorly, was greatly thickened, as a result of great increase in the thickness of the submucous coat. The mucosa was normal. The serous coat was smooth and free from adhesions. Metastases were absent. Histologically, the growth was regarded as an endothelioma arising from the lymphatic spaces of the submucosa.

Brooks[15] records three further cases of sarcoma of the stomach, and emphasises the following as differentiating it from carcinoma. The sarcoma tends less to involve the mucosa, and more to traverse the muscular coat. Metastases are less frequent, and appear later than in carcinoma. The duration of the disease is greater. The stomach contents show free hydrochloric acid and an absence of blood. Surgically treated cases of sarcoma afford a prognosis better than cases of carcinoma.

With regard to *cancer* of the stomach, one of the most interesting communications is that of Moore[16] and his co-workers, in which they endeavour to solve the much-discussed question of the cause of the disappearance of hydrochloric acid from the gastric secretion. The prevalent view, that it depends on a co-existing gastritis, or on neutralisation of the acid by alkaline fluid exuded into the stomach from the ulcerated surface of the growth, is untenable, as the condition obtains in the absence of both gastritis and ulceration. Examination of the stomach contents after a test-breakfast, in 17 cases of malignant disease of organs other than the stomach—uterus, mammary gland, prostate, colon, mouth, etc.—revealed absence of free hydrochloric acid in two-thirds, and its marked diminution in the remainder. The investigators accordingly sought the cause in some more general condition, viz., in some alteration of the blood and tissue fluids, either secondary to and induced by the presence of the cancer cells in the organism, or primary and made manifest by the diminished production of hydrochloric acid, and by the cellular proliferation constituting the new growth.

The former hypothesis was at once negatived by the fact that extirpation of the growth had no effect in restoring the hydrochloric acid secretion, and it remained that both the achlorhydria and the neoplastic process might have a common basis in an alteration of the alkalinity of the tissue fluids, not as expressed in the degree of their acid-combining power, but in the relative concentration of H ions and OH ions in these fluids.

The production of hydrochloric acid by the cells of the gastric mucosa can happen only when the H ions predominate in the blood; with their relative diminution and increase in the OH ions, the production of hydrochloric acid ceases, and the alkalinity of the blood increases. The investigations of Loeb have demonstrated that a minimal increase in the alkalinity of the fluid, in which cells are living, suffices to stimulate them to excessive proliferation and growth.

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* * *

SOME FAMOUS QUACKS.

VI.

JAMES GRAHAM.

CARLYLE somewhere says that, by careful search, it might be possible to discover the greatest fool extant at any given time. It might be difficult to say who was the greatest quack that ever lived, but if matchless impudence were to be accepted as the test, a well-founded claim might be made on behalf of James Graham, the high priest of the Temple of Health of which the Goddess was Lady Hamilton, originally Emma Lyon, that Emma whom Nelson recommended to the care of the country for which he died. Thither at one time flocked the fashionable society of London, to listen to what many of them must have known to be quackery of the vilest kind, and to be treated by methods openly intended for the stimulation of lust rather than for the healing of disease. Graham was born at Edinburgh in 1745; he was the son of a saddler in the Cowgate. He studied medicine in the University of his native city under Monro Primus, Cullen, and Black, but it is doubtful whether he ever took a degree or obtained a qualification of any kind. In those days, however, many honest practitioners looked upon a diploma as a needless luxury. After fleshing his lancet at Pontefract, where he married in 1770, he sought his fortune as an itinerant oculist and aurist in America. He spent some two years in Philadelphia, where he became acquainted with Franklin's discoveries in electricity, of which he had the wit to see the possibilities as a means of exploiting the folly of the public. In 1774 we find him at Bristol, advertising the wonderful cures he had wrought. After spending a short time in Bath, he established himself in London in 1775. In 1777, however, he was back at Bath attracting people to his net by promises of cure with his "ætherial" and "balsamic" medicines, his milk baths, dry friction and electricity. His confiding patients were made to sit on a "magnetic throne" or were placed in a bath, into which electric currents could be passed. He became fashionable and aspired to a larger sphere. In 1779 he was in Paris, where he met Franklin, and at Aix-la-Chapelle, where he received high testimonials from many aristocratic patients, including Georgiana, the famous Duchess of Devonshire. Later in the same year he opened the Temple of Health on the Royal Terrace, Adelphi, facing the Thames. His house and "plant"—the word is singularly appropriate—are said to have cost at least £10,000. There for a time Graham flourished exceedingly. There he lectured, made a show of his house and sold his nostrums. The entrance hall was decorated with crutches and other disused accessories of invalidism. A description of the Temple of Health may be found in the *Chronique Scandaleuse.* The entablature was decorated with figures of Venus, Minerva, and Juno. Lower down was a statue of Æsculapius, and on the door was an inscription to the effect that it was not guarded in order that the poor might be as free to enter as the rich. Nevertheless, two gigantic porters played the part of twin Cerberi at the gate; they were dressed in flowing robes over which they wore coats of mail inscribed with the words "Temple of Health," and they allowed no one to pass till a fee of £6 had been paid. Having gone through this rite of initiation the visitor was admitted. As soon as he set foot on the stairs the strains of a hidden band were wafted to his ears through small openings in the steps, and the perfumes of Araby breathed around him. In time he came to the "Great Apollo apartment," a magnificent room lavishly decorated with festoons, gilt and silver ornaments, and a number of mirrors reflecting innumerable lights. There every afternoon from five till seven the high priest of the shrine delivered his lecture, which was always preceded by music. After the lecture the audience received electrical shocks from conductors hidden underneath covers of the seats. Their minds thus prepared, a gigantic spectre came up through the floor and handed a bottle containing liquid, presumably the "ætherial balsam," to the doctor. Then came a handsome woman, representing the Goddess of Music, who brought the performance to a close with song. Then various afflictions were treated by electricity. There was also a High Priestess at the Temple who read lectures to ladies, at which Lady Hamilton, "the rosy, athletic and truly gigantic Goddess of Health and Hymen" posed on the "celestial throne."

Graham's most dainty device for the extraction of money from the public was his "celestial bed." This was the inner mystery of his establishment to which all the rest were but accessories. The bed was a gorgeous structure made by Denton, a tinman of great mechanical skill. Graham himself describes this invention in a *Lecture on Love*, containing, as he said, salutary advice of such consequence to all mankind that he declared himself prepared to "deliver it to each who chooses to accept it at the very moderate price of one guinea." After setting forth the fertilizing virtues of his "divine balsam"—"a preparation of rich gum with either æther, electricity, air or magnetism!"—he goes on to say:

"Should pregnancy at any time not happily ensue, I have a most astonishing method to recommend, which will infallibly produce a general and happy issue, I mean my celestial or magnetico-electrical bed, which is the first and only one that ever was in the world; it is placed in a spacious room to the right of my orchestra, which produces the celestial fire and vivifying influence; this brilliant celestial bed is supported by six massive glass pillars, with Saxon blue and purple satin, with Arabian spices in the style of those in the seraglio of the Grand Turk. Any gentleman and his lady desirous of progeny, and wishing to spend an evening in this celestial apartment, after c———n, may by a compliment of a £50 banknote, be permitted to partake of the heavenly joys it affords, by causing immediate conception, accompanied with soft music; the superior pleasure and ecstacy which the parties enjoy in the celestial bed is really astonishing, and never before thought of in this world; the barren must certainly become fruitful when they are powerfully agitated in the delights of love."

This wonderful invention, he says elsewhere, was the fruit of his acquaintance with Franklin's discoveries in electricity:

"It was in Philadelphia, the capital city of the infant queen of empires, where I had the good fortune of studying electricity two years, profiting by the labours of the great prince of philosophers!—and (might I not have added)—of the venerable prince of politicians. Five years, gentlemen, had I the happiness of sojurning in that boundless land, which then overflowed with abundance, which flowed with liberality and whose rising sun announced the great day of Universal Freedom and shone with intellectual light! During my residence in that wise and all-tolerating city, I was suddenly struck with the thought, that the pleasure of the venereal act might be exalted or rendered more intense, if performed under the glowing, accelerating, and most genial influences of that heaven-born, all-animating element of principle, the electrical or concocted fire! At that time, young, enterprising, impatient, and eccentric, I speedily insulated (or raised on strong glass pillars) a common bedstead, and filled it with copious streams of the electrical fire, conveyed by metal rods, inclosed in glass tubes, through the partition wall from the adjacent room where the great globes were wrought,—globes, I say, for, at that time, we had few, or no cylinders in America, for exciting the fire. I recommended the trial of this, then whimsical, bed to several of my medical, philosophical, and gay friends. They all found that my theory had been founded on nature, that it was even capable of demonstration; and that the pleasure was, under those circumstances, rendered not only infinitely more intense, but, at the same time, infinitely more durable! insomuch that, after a few months, when they were merry over a glass, and when it had circulated so freely as to make them forget themselves and depart from that delicacy and reservedness which ought ever to be observed in these matters, they talked, not as other men might have done, of the happy minute, or of the critical moment, no! they talked comparatively of the critical hour! ! !"

He adds that he learnt the "vivifying prolific quality of the electric fire" from the case of "a good stout-like Dutch woman" who for two years had been paralysed and suffered from menstrual obstruction, and who by sleeping in the electric bed was not only cured of those infirmities but became "the healthy and happy mother of a pretty large family of children."

It may easily be conceived that Graham, for a time at least, drove a roaring trade. He is described by Brissot de Warville, a French politician who sought shelter in London from the inconvenient attentions of the French Government, and who afterwards was a victim of the guillotine, in his

Correspondance Secrète, as a man of imposing presence with a fine head. In Paris all the women on the boulevards turned to look after him. He lectured with the solemnity of a pontiff. Of the matter of his lecture an idea may be formed from the specimens that have been given. But there is much besides that it would be impossible to print.

It is only fair to say that mixed up with obscene quackery, occasionally tinged with blasphemy to make it more piquant, Graham gives a good deal of sound advice as to personal cleanliness and hygiene which was greatly needed at the time. He may be called a prophet of the open window. He says :—

"Sleep on a hair mattress, or rather on a bed of wheat or oat straw, with the grain in the ears of the corn, and with all the windows of your room wide open. This is my own invariable custom, summer and winter, even in the wettest, coldest and stormiest weather ; and I never, never have a cold, low spirits or any indisposition whatever. The free open cold air is a constant full cold bath for the outside of the body, for the lungs, for the mass of blood which passes through them, and consequently for the whole system, of the friendliest and most efficacious kind."

Elsewhere he says that "open air day and night" and "full exercise in the free open air" with cleanliness, are "the grand cosmetics ; the universal beautifiers ; the infallible preservers of health and the prolongers of human life to the very longest period of human existence ; I mean to the age of a hundred and fifty, stout, healthy, and happy years." But the innate quackery of the man always pierces through the veneer of good sense with which he occasionally strove to cover his moral nakedness. He goes on to say :—

"I do myself expect that by a constant attention to all the above particulars, *and by the daily use of a certain balmy rejuvenating medicine*, which I had the supreme blessedness of discovering many years ago and which I have now brought to a high degree of perfection, to live in perfect health till I shall be at least a hundred and fifty years old, and to be, when a hundred years of age, as robust, as healthy, fresh, active, and younglike as men in general are, in these degenerative and luxurious times at fifty."

It must be accounted to him for righteousness that he preached frugality and moderation in diet, and denounced alcoholic excess, at a time when gross feeding and drunkenness were all but universal. He held that all diseases were caused by wearing too much clothing, and he himself wore no woollen garments. Graham in his later days was an advocate of mud baths. He is said to have remained naked in the earth for several hours on nine days in succession. There is an account of him and a young lady at Newcastle stripping "into their first suits" and being interred up to the chin, their heads beautifully dressed and powdered, appearing not unlike two fine full-grown cauliflowers. Robert Southey twice saw Graham, whom he calls "half knave, half enthusiast," in his mud bath.

His vogue in London did not last long. Horace Walpole, who went to hear him in 1780, describes him as "the most impudent puppet-show of imposition I ever saw, and the mountebank, himself the dullest of his profession, except that he makes the spectators pay a crown apiece." He was satirized on the stage, and in 1782 he was sold up. In 1783 we find him lecturing at Edinburgh. There he soon came into collision with the civil power and was imprisoned for his "injurious publications." He issued a counterblast entitled "A Full Circumstantial and most Candid State of Dr. Graham's Case, giving an account of Proceedings, Persecutions, and Imprisonments, more cruel and more shocking to the laws of both God and man than any of those on record of the Portuguese Inquisition." When he came out of gaol he began lecturing again, but was fined and soon sought pastures new in various towns where he met with the like vicissitudes of fortune.

In his declining years he turned to religion. He had preached to his fellow prisoners in the Tolbooth of Edinburgh, and, in 1789, he told the public of Bath that he "regretted the extravagances of youth and a warm imagination uncurbed by Christianity, but was now passing into 'the mild serenity of an evening natural, and of an autumn intellectual sun.'" This phase seems to have marked a period of transition to insanity. Southey says that at this time he "would madden himself with opium, rush into the streets and strip himself to clothe the first beggar he met." Graham retired to Edinburgh, where he was for some time confined in his own house as a lunatic. His last pamphlet opens with an affidavit made on April 3, 1793, that from the last day of December 1792 to January 15, 1793, he neither ate, drank, nor took anything but cold water, sustaining life by wearing cut-up turfs against his body, and by rubbing his limbs with his own nervous æthereal balsam. This recalls Morrison taking his own pills to the last gasp. Graham's balsam did not prevent his dying suddenly at Edinburgh in 1794, thus falling short of his boasted expectancy of life by a century.

In the following list of Graham's publications, given by Bettany in the *Dictionary of National Biography*, are mirrored the various phases of his diversified career :—

1. "An Address to the Inhabitants of Great Britain, particularly to those residing in the Great Metropolis of the British Empire," containing his professions and promises as an oculist and aurist, with accounts of cures in America, Bristol, and Bath ; London, 1775. 2. "The Present State of Practice in Diseases of the Eye and Ear," 1775. 3. "A Short Inquiry into the Present State of Medical Practice in Consumptions, Asthmas," &c. ; London, 1776. 4. "The General State of Medical and Chirurgical Practice exhibited, showing them to be inadequate, ineffectual, absurd, and ridiculous"; 6th edition ; Bath, 1778. This book contains "The Christian's Universal Prayer," composed by Graham. 5. "A Treatise on the All-Cleansing, All-Healing, and All-Invigorating Qualities of the Simple Earth, when long and repeatedly applied to the Human Body, &c. . . ." London, 1779. 6. "A clear, full, and faithful Portraiture . . . of a certain most beautiful and spotless Virgin Princess to a certain Youthful Heir-Apparent." London, 1779 ; dedicated to the Prince of Wales, and recommending merely the "Wisdom of Solomon." 7. "A Sketch or Short Description of Dr. Graham's Medical Apparatus, erected about the beginning of the year 1780, in his house on the Royal Terrace, Adelphi"; pp. 92 ; London, 1780. An appendix contains a description of his three great medicines : the electrical ether, the nervous æthereal balsam, and the imperial pills ; London, 1782. 8. "The Guardian Goddess of Health," n.d. (1780–3). 9. "Il Convito Amoroso, or a Serio-Comico-Philosophical Lecture on the Causes, Nature, and

Effect of Love and Beauty . . . as delivered by Hebe Vestina at the Temple of Hymen," 1782." 10. "A Discourse delivered on Sunday, 17 August 1783, to his fellow prisoners in the Tolbooth of Edinburgh on the Resurrection of the Body and the Immortality of the Soul," 1783. 11. "An Appeal to the Public, containing the full account of the ignorant, illegal, and impotent Proceedings of the contemptible Magistrates of Edinburgh. . . ." 1783. 12. "A Lecture, on the Generation, Increase and Improvement of the Human Species . . ." 4to, pp. vi and 22 in small type, 1783. 13. "Travels and Voyages in Scotland, England, and Ireland, &c.," 1783. 14. "The Principal Grounds, Basis, Argument, or Soul of the New Celestial Curtain (or Reprehensory) Lecture . . . London, 1786. (On page 3 Graham describes himself "Formerly a Physician, but now a Christian Philosopher.") 15. "Proposals for the Establishment of a New and True Christian Church," Bath, 1788. 16. "A New, Plain, and Rational Treatise on . . . the Bath Waters," Bath, 1789. 17. "A Short Treatise on the All-Cleansing, All-Healing, and All-Invigorating Qualities of the Simple Earth," Newcastle-upon-Tyne, 1790 (a new pamphlet). 18. "The Guardian of Health, Long Life, and Happiness," etc., Newcastle, 1790. 19. "Address to the Diseased, Weak, and Lame," Newcastle, 1790. 20. "A New and Curious Treatise on the Nature and Effects of Simple Earth, Water, and Air when applied to the Human Body; How to live for many weeks, months, or years without Eating anything whatever; with the Extraordinary Histories of many Heroes, Male and Female, who have so subsisted," London, 1793.

Reviews of Books.

A Manual of Pharmacology. By WALTER E. DIXON, M.A. (Cantab.), M.D., B.Sc. (Lond.), D.P.H. Camb., etc., Professor of Pharmacology in King's College, London; Assistant to the Downing Professor of Medicine in the University of Cambridge. Pp. 451. Illustrated. London: Edward Arnold. 15s. net.

As Professor Dixon states in his preface, this is not a text-book on materia medica, at the same time we consider that it deals very fully with the rational and experimental side of the materia medica. Pharmacology is indeed an experimental science, and, although, for reasons readily understood, the experimental method cannot be adopted on man, except in a very limited number of cases, yet incalculable benefit has been obtained from our knowledge of the action of drugs, which has been derived from experiments upon the lower animals. "Pharmacological research has for its object the diminution of human suffering and the prolongation of life." We highly commend this *scientific* work to all practitioners of medicine, who take an interest in the action of drugs, and a rational view of prescribing medicine. The book is well illustrated, and the method of dealing with the action of drugs original.

Recent Advances in Physiology and Bio-Chemistry. Edited by LEONARD HILL, M.B., F.R.S. Pp. 740, with diagrams. London: Edward Arnold. 18s. net.

IN his preface, the Editor points out that, in the text-books, all branches of physiology are treated as of equal value; but that in reality many parts of the science have little bearing on practical medicine, whilst other portions are of fundamental importance, and cannot be studied too deeply. The object of the present work is to extend those portions of physiology, which are of paramount importance to the practical physician and surgeon, and in the endeavour to do this, the Editor has been greatly helped by the other contributors, viz., Drs. Beddard, Macleod, Moore, and Pembrey. Such subjects as Diabetes, Uric Acid Metabolism, Hæmolysins and Immunity, Mountain Sickness, Caisson Sickness, the Metabolism of Fat and the rational treatment of Obesity; the influence of temperature and the relative dryness of the atmosphere, of work, diet, baths, clothing, etc., upon metabolism are very fully treated of. The causation of dyspnœa, of Cheyne-Stokes respiration, the influence of the thyroid and supra-renal glands upon metabolism are carefully considered. This excellent work, which is admirably produced, may be recommended to all medical men, who desire to acquaint themselves with the latest views upon the subjects mentioned above. Moreover, the book should be carefully studied by all senior students in physiology who intend to present themselves for any of the higher examinations.

On Carbohydrate Metabolism, with an Appendix on the Assimilation of Carbohydrate into Proteid and Fat, followed by the Fundamental Principles and the Treatment of Diabetes dialectically discussed. By F. W. Pavy, M.D., F.R.S., Consulting Physician to Guy's Hospital. London: J. & A. Churchill. 6s.

This work embodies a course of advanced lectures in physiology delivered at the London University with two added appendices. In his lectures, Dr. Pavy details experiments in support of his well-known contention that free sugar cannot exist in the blood without flowing off in the urine, and shows that a certain proportion of sugar is carried and excreted in a glucoside form. As the outcome of his latest researches, described in the appendix, the author assigns to the lymphocytes present in the intestinal villi the function of incorporating the digestive product glucose as a glucoside component of proteid, which they thus convey to the tissues viâ the lacteals and blood. An analogy is thus established between the assimilation of proteid and carbohydrate, since the products of digestion (peptone and dextrose), do not appear as such in the blood. Many other observations are brought forward to support this theory, which, despite its weak points, is consistent and offers valuable suggestions.

The treatment of diabetes is fortunately independent of the acceptance, or rejection, of the glycogenic doctrine. Dr. Pavy holds the ordinary principles, and is sceptical as to the value of the "potato cure." He rightly regards the absolute arrest of glycosuria as the only rational aim of the physician, and deprecates any lowering of this standard.

Diabetes Mellitus: its Pathological Chemistry and Treatment. By Professor von Noorden, translated by Florence Buchanan, D.Sc., and Walker Hall, M.D. Bristol: John Wright & Co. 5s.

In this latest addition to his well-known treatises on disorders of metabolism, Professor von Noorden has made a masterly contribution to the study and treatment of diabetes. In spite of the difficulties of so intricate a subject, the author's account is particularly clear and easy to follow, since it avoids all abstruse theoretical discussions and unnecessary recapitulation of cases.

The book consists of two parts; the first describes in detail the pathological chemistry of the disease, the second applies this knowledge to scientific treatment; symptomatology is purposely omitted. One of the most interesting chapters deals with the formation of acetone bodies and their relation to carbohydrate-free diet—facts which are not yet fully known or described in text-books. The section on treatment is full of useful information, and insists on the careful study of individual cases with regard to their powers of carbohydrate assimilation. The employment of drugs, of Carlsbad treatment, and the value of the various special "cures" are impartially presented in the light of extensive experience. A useful appendix gives the carbohydrate values of different foods compared with the standard of white bread: hence if a diabetic is limited to 120 gms. of bread per diem, it is easy to calculate the amount of fruit, etc., by which part, or the whole, may be replaced.

That it is a translation is almost unnoticeable, and the book is one to be cordially recommended as an excellent guide.

Modern Surgical Technique in its relation to Operations and Wound Treatment. By C. YELVERTON PEARSON, M.D., M.Ch., F.R.C.S.; Professor of Surgery, Queen's College, Cork, etc., etc. Pp. 371, and index. Two coloured and other plates, and 111 illustrations in the text. London: John Bale, Sons & Danielsson, Ltd. 10s. 6d. net.

THE greater part of this volume is occupied by a clear account of the methods, in present use, for the prevention of the introduction of septic organisms into a surgical wound.

Of antiseptics: biniodide of mercury and solutions of iodine are highly praised, the latter being recommended especially in cases of chronic suppuration.

The Claudius iodine method for the preparation of catgut for ligatures and sutures is advocated as being both simple and safe, though in situations where greater stability is required the use of formalinised gut is advised.

The question of the use of gloves, either of rubber or of cotton, is discussed, and the opinions of various authorities quoted; the surgeon being ultimately advised to make his own decision on the point.

The subjects of the arrest of hæmorrhage, the closure of wounds, and the treatment of the patient before and after operation, receive due attention; and a space necessarily small, is devoted to the arrangement of the operating room. The general teaching is that of the "aseptic" school of surgery, and the methods of the "antiseptic" surgeon are but lightly touched upon.

In view of the number of similar works extant, it is a little difficult to appreciate the need for that now before us. It is, however, of handy size, concise and accurate, and is perhaps as likely to be of as much value to the operating surgeon, as any other work of like scope with which we are acquainted.

A Treatise on Surgery. By GEORGE RYERSON FOWLER, M.D., Examiner in Surgery, Board of Medical Examiners of the Regents of the University of the State of New York; Emeritus Professor of Surgery in the New York Polyclinic; Surgeon-in-Chief to the Brooklyn Hospital, etc., etc. Vol. I. Pp. 685 and index; with 888 text illustrations and 4 coloured plates, all original. London: W. B. Saunders & Co.

WE must confess to some degree of disappointment which we have experienced in the perusal of this treatise on surgery.

The first volume deals with general surgery of the body, and with the regional surgery of the head, neck, and thorax. To these are added chapters on inflammation, anæsthesia, and laboratory aids to diagnosis.

We need hardly point out the difficulty of dealing with such an extensive programme within the small limits of this volume, especially when the very large number of illustrations and the large type of the letterpress are also considered. The result is, as might be expected, that many subjects suffer from undue compression, and that, in particular, differential diagnosis and after-treatment receive but scant attention.

The illustrations are particularly good, though we think that many are somewhat unnecessary, as, for instance, those depicting various instruments and bandages which seem to us to occupy much space which might have been put to better use.

The chapter on laboratory aids is especially good, and we think that

Professor Fowler has been well advised in keeping it down to short length, the temptation to elaborate such a chapter must have been great.

The diagnosis and treatment of injuries and diseases of the brain occupy only 20 pages, which is a typical instance of the way in which space has been economised. On the other hand, 12 pages are allotted to the subject of carcinoma of the breast, nearly half of this being occupied by illustrations.

The whole work seems to us somewhat unequal, and we may perhaps be allowed to enter a protest against its excessive weight, which is, we believe, due to the free use of bismuth in the preparation of the paper.

A Manual of Midwifery for Students and Practitioners. By HENRY JELLETT, B.A., M.D. (Dub. Univ.), F.R.C.P.I., L.M., Gynæcologist and Obstetric Physician to Dr. Steevens' Hospital, Dublin; Extern Examiner in Midwifery, Royal University of Ireland; Examiner in Midwifery, Royal College of Physicians, Ireland; Ex-Assistant Master, Rotunda Hospital; Ex University Examiner in Midwifery and Gynæcology, Dublin University. Pp. xxv + 1158. 467 plain and coloured illustrations and 9 plates. Demy 8vo. London: Baillière, Tindall & Cox. 21s. net.

This book is the work of several writers, the author having secured the assistance of certain gentlemen whom he considered especially qualified to deal with those subjects requiring a special knowledge of Anatomy, General Medicine, Pathology, and Mental Diseases. Thus Dr. T. G. Moorhead has contributed the chapters on Embryology and Anatomy, and the sections on the Phenomena of Pregnancy and on the Anatomy of Contracted Pelvis; Dr. H. C. Drury the chapters on Infectious Diseases, and on Organic and Functional Diseases of Pregnancy; Dr. R. J. Rowlette the section on the Ætiology and Pathology of the Surgical Fevers of the Puerperium; and Dr. W. R. Dawson the sections on the Insanities of Reproduction.

The special knowledge which has been brought to bear on these subjects has been cleverly distributed, and has not interfered with the general harmony of the book, which is divided into ten parts. Part I. deals with Obstetrical Anatomy, Maternal and Ovular, and Part II. with the important subject of Asepsis and Antisepsis, included in which is a full description of the Bacteriology of the Genital Tract. The next six Parts are devoted to the full consideration of the Physiology and Pathology of Pregnancy, Labour, and the Puerperium. Part IX. to the various Obstetrical operations. The concluding chapter concerns the Infant, its Physiology and Pathology.

In the letterpress under the figure 224, the right-hand "on" is evidently a misprint for "in" the uterus, and again, in the description of Anterior Fontanelle Presentation, there is an obvious error in stating that "the head engages with its occipito-frontal diameter in the antero-posterior diameter of the pelvis."

This book deserves to find high favour, and will certainly hold its own with the numerous other works on the same subject. The illustrations are numerous, and in many cases original.

* * *

NOTES FROM FOREIGN JOURNALS.

TREATMENT OF ŒDEMA OF THE LUNGS.

A very full discussion followed the reading of a paper on this subject by Reisman of Philadelphia, at a recent meeting of the Association of American Physicians in Washington. The remedies mostly depended upon proved to be atropine, strychnine, morphia, and adrenalin. With regard to the last named, Meltzer, of New York, pointed out that its use, even if apparently successful in some cases, had no rational basis to depend upon, for it stimulates the peripheral blood-vessels and raises arterial pressure throughout the systemic circulation. This stimulation does not extend to the blood-vessels in the lungs, so that the pulmonary engorgement is increased as the blood passes from the high pressure of the systemic circulation to the low pressure of the pulmonary circulation Meltzer, therefore, urges an opposite method altogether, using hypodermic injections of nitro-glycerine, which drug relaxes the peripheral circulation and so tends to diminish the pulmonary congestion. Recent experiments in England and in France, have shown that the use of nitrites, in checking hæmorrhage from the lungs, is due to the fact that these vaso-dilators produce an actual constriction of the vessels of the lung. If the lung of an animal is exposed, and nitrite of amyl is administered, the lung-tissue is seen to become blanched simultaneously with the fall of the blood pressure in the general arterial system.

Œdema of the lungs may be due to a variety of causes, a fact which explains the varying degrees of success, following the use of different drugs. In urgent cases there was a consensus of opinion that morphia and nitroglycerine should be given freely. In cases of more gradual development, in which the general arterial tension is low, atropine may be freely administered, as much as ^{1} th of a grain being given, and strychnine may be used.

H. A. Hare reminded the meeting that, as œdema of the lungs is a symptom rather than a disease, treatment must depend upon a careful study of the patient's general condition. When arterial spasm is present, nitro-glycerine is invaluable; but, when tension is very low, atropine and strychnine must be employed.—(*Therapeutic Gazette.*)

TREATMENT OF GONORRHŒAL RHEUMATISM.

A clinical lecture on this subject, by Professor Albert Robin, is fully reported in the *Revue de thérapeutique médico-chirurgicale* of December 1st and 15th. Robin considers the disease to be one for medical treatment only, and rejects the idea of surgical intervention by opening up the joint at an early stage, which has been recommended. It is, however, impossible to lay down any hard-and-fast rule for treatment, each case requires to be treated on its merits. To show the necessity for this, he dealt, in his lecture, with three cases under treatment in the Hôpital Beaujon. The first was that of an hotel waiter, 28 years of age, with a history of a severe attack of acute rheumatism ten years previously. Since this attack he had frequently suffered from pains in the joints. He had a first attack of gonorrhœa two years ago, and suffered, at the same time, from gonorrhœal conjunctivitis, the result of direct infection. In October of this year, he developed gonorrhœa, three days after exposure to contagion, and a week later, was seized with acute rheumatism, affecting most of his joints, but later the disease was limited to the joints of the left leg and foot and the left thumb. Two days after the rheumatism appeared, intense conjunctivitis, not due to direct infection, affected both eyes.

The second case was that of a man, of sixty years of age, the subject of a recent gonorrhœa, who, ten days after the urethritis appeared, was seized with fever and acute rheumatism, affecting all the joints of the left lower limb. A fortnight later, the pains left the leg and settled in the right hand, and symptoms of cystitis appeared, as well as signs that an ascending pyelo-nephritis was present. Later the testicles were implicated.

The third case was that of a woman, aged thirty-five years, in whom the disease had settled in the right upper limb, leaving the arm powerless, the wrist half ankylosed and fingers quite rigid, the shoulder affected, and the elbow immovable. The muscles of the forearm and the interosseal muscles were atrophied.

The first case was treated with salicylate of soda (gr. viiss 3^{tis} horis), and in two days, all pains and swellings, had gone, except in the right ankle, which Robin considered was the only joint really involved in the gonorrhœal affection. He recommends the use of salicylate in those cases of gonorrhœal rheumatism in which several joints are affected, because of its analgesic effects. The urethral discharge should be treated, but neither by antiseptic injections, nor balsams. Robin prefers to let the discharge go on and cure itself, an old-fashioned idea, which, however, is now found to be based on good principles, since the gonococcus eventually kills itself by its own free growth. In the case under consideration, the discharge had ceased when the rheumatism began. The patient was given two litres of a decoction of fir-buds to drink in the twenty-four hours, to which was added about one drachm of bicarbonate of soda to correct the acidity of the urine. A creamy purulent discharge soon reappeared in abundance, gradually decreased, and, after a few days, altogether stopped.

The conjunctivitis was treated wlth an old remedy now almost forgotten.

℞.	Cupri Sulph. - - - -	gr. iss.
	Tinct. Opii - - - - -	m. x.
	Aq. Destill. - - - -	ad. ℥j.

Misce. Fiat collyrium.

"One drop to be instilled twice a day over the lachrymal caruncle." In addition, the eyes were washed out twice a day with boric acid lotion.

The arthritis in the foot had become sub-acute : the treatment of this will be dealt with later.

In the second case, the condition of the kidneys forbade the use of salicylate of soda, but, whether real nephritis was present or no, the treatment was the same, viz., milk diet and disinfection of the urinary passages. The patient was given the decoction of fir-buds and urotropin, the latter in $7\frac{1}{2}$ grain doses three times a day. Robin finds this dose sufficient, as a rule, but, if the stomach tolerates the drug, he increases the dose to $12\frac{1}{2}$ grains. If there is any doubt as to the complete acidity of the urine, this can be ensured by giving small doses of benzoate of soda. In the case under consideration, the pyelitis and cystitis cleared off rapidly under this treatment. When he finds that the pyelitis does not yield to this treatment, he makes use of another forgotten remedy, the oil of Haarlem, a mixture of essence of juniper berries and oil of bays, giving 10 to 15 drops emulsified in syrup of Canada balsam or in decoction of soap-bark. As a last resort, turpentine and camphor should be given in pills.

℞. Terebinthinae Venetae	ʒiiss.
Camphoræ	ʒiss.
Ext. Opii	gr. iv.
Ext. Aconiti.	gr. iiss.

Misce. Ft. pil lx.

"From two to six to be taken in the 24 hours."

The local treatment of the arthritis consists of immobilisation of the joint, which relieves the pain, and possibly checks the inflammatory condition of the joint, but the fixation of the joint must be compassed without compression by bandages, only using a light splint, well padded, and with sufficient openings to allow observation of the joint, and the application of local remedies. Robin prefers for the latter a liniment recommended by Bourget of Lausanne.

℞. Acid. Salicylici	ʒiiss.
Ess. Terebinthinæ	ʒiij.
Adipis	℥iiss.
Lanolini	℥iiss.

Misce.

If this does not succeed he applies wet compresses of Van Swieten's fluid (corrosive sublimate part 1, alcohol parts 100, water parts 899).

Immobilisation of the joint must be as short as possible, in view of the danger of ankylosis. As soon as the joint ceases to be painful, the limb must be taken out of the splint, light massage (effleurage) begun, and a certain amount of careful passive movements made.

He does not commend Bier's method, but has been encouraged to make a further trial of metallic ferments as he has had good results from the use of colloidal palladium, given by subcutaneous injection in doses of 1 to 3 hundredths of a milligramme per cubic centimetre. Collargol, used as an ointment, has always been satisfactory. The radio-carpal joint of the second patient mentioned was submitted to the action of radium, and the pain disappeared most remarkably quickly; but Robin points out that this was just at the time when the orchitis developed. However, he attributes the quite exceptional quickness with which the pain disappeared to the action of the radium.

In the third patient, the chronic stage of the affection had been reached, and other treatment than the foregoing was necessary. She still had a slight discharge. This required to be stopped and not encouraged as in the other cases. Injections of 1 in 1,000 of permanganate of potash, or ¼ per cent. of protargol, were used locally; internally the patient was given another old-time remedy.

℞. Balsam. Copaibæ	ʒv.
Pulv. Cubebæ	ʒx.
Ferri Tartarati	gr. xlv.
Magnesiæ	ʒiiss.
Aq. destill.	ʒiss.
Syrupi Cydoniæ	q.s.

Misce.

"Take every day 5 or 6 boluses the size of a hazel-nut."

For the ankylosis and muscular atrophy, massage must be the first means employed, and it must be directed, not only to the restoration of the mobility of the joints, but also to the revival of the vitality of the tissues and muscles, as well as the re-education of such muscles as the patient is still able to use, since these must now supply the place of those whose function has been lost.

Electricity may be used in continuous currents of high intensity applied directly to the joint, a method which Robin considers rather dangerous; or preferably in faradic currents of low intensity acting on the different groups of muscles, especially those distant from the joint.

Counter-irritation is useful, if obtained by means of applications of tincture of iodine and small superficial cauterisations frequently repeated. The "turpentine bath" is very good, as are also hot baths, hot sand-baths (70° C. and rising by degrees to 80°), and local applications of hot air (Tallermann's method). All these help in the re-absorption of exudation, and hasten the reduction of the ankylosis.

Finally, the general condition of the patient must not be overlooked. As soon as the discharge is completely cured, the administration of iron must be continued. (Syrup. Ferri Cit. ℥ss. bis die post cib.). After ten days, this must be alternated with—

℞. Sodii Arseniat.	gr. $\frac{4}{5}$
Potass. Iodidi.	gr. lxxv.
Aq. destill.	℥x. M.

"Take one tablespoonful in the early morning, and one before the mid-day meal for ten days."

In conclusion, Robin urged that aspiration of the joint, and washing it out with an antiseptic injection, should only be resorted to when the swelling and pain do not yield to any of the methods he had pointed out. He reiterated his condemnation of arthrotomy, except in the case of suppuration supervening in the articulation.

SOME CONSIDERATIONS IN THE TREATMENT OF TUBERCULAR PHTHISIS.

A leading article, in the *Therapeutic Gazette* for November, very wisely points out that there are other organs than the lungs to be considered in the treatment of pulmonary tuberculosis. The administration of creosote and similar substances, with the object of destroying the tubercle bacillus in the lungs is quite useless, and is based upon a wrong idea of the pathological processes present. The body tries to cope with the disease by surrounding the infected area with a zone of exudation, and thus cuts off the bacillus from any substances circulating in the blood. Consequently, any body, powerful enough to kill the bacillus in the tissues, must of necessity do serious damage to the tissues. Even drugs, which do some good in certain directions, may do a great deal more harm in the course of elimination by irritating organs already impaired in function by the presence of the disease elsewhere. Again, drugs given for the purpose of combating the infection very frequently upset the digestion, or cause loss of appetite, whereby the patient's ability to struggle with his disease is weakened. It is, therefore, important to bear in mind the condition of the stomach, bowels, and kidneys, when tubercle in any part of the body is being treated medicinally. According to Heyn, a very large proportion of tubercular patients have distinct lesions in the kidneys, either some degree of nephritis, or actual invasion by the tubercle bacillus. This is supported by Walsh, who found that, out of 33 autopsies, tubercles were present in the kidneys in 58 per cent. In some cases, the nephritis is probably due to the tubercular toxin rather than to the bacilli. These facts emphasise the necessity for extreme caution in giving drugs like creosote and the iodine preparations, which are known to irritate the kidneys.

SILVERED-SILK LIGATURES.

Witzel and Wederhake recently described, in the *Zentralblatt fur Chirurgie*, a method of treating silk for ligatures, which renders it non-absorbent and non-absorbable, smooth, strong, and germicidal, thus obtaining some of the best qualities of silver wire without loss of flexibility, and providing a most efficient material for buried sutures. The following are the brief details of preparation :—

1. The silk is wrapped round glass plates, and freed from fat by soaking in ether for twelve hours, and then in absolute alcohol for twelve hours.

2. It is then soaked for twenty minutes in 10 per cent. hydrogen peroxide solution.

3. It is then placed in the silver solution for one hour. This solution consists of a 1 per cent. solution of nitrate of silver, in which the silver has been completely precipitated by sodium hydrate, and the precipitate re-dissolved in ammonia, no more of the latter being used than is necessary for solution. The silk, on removal from this solution, ought to be quite black and polished, showing no white spots on cross-section.

4. It is then dried in an oven at 100° C.

5. It is then placed in pure chloroform for two hours, and from this transferred to a solution of pure rubber in chloroform (16 per cent.), where it remains for twelve hours.

6. On removal it is quickly washed in chloroform, dried, and then boiled for ten minutes in 1 per cent. sublimate solution.

It is claimed that silk, so treated, is stronger by one-third than ordinary silk, is sterile, absorbs no water, and is perfectly flexible. The cost of preparation is said to be inexpensive.

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Notes by the Way.

The Royal Society of Medicine.

THE fusion of the medical societies of London into one strong body that should be able, like the French Académie de Médecine, to speak with authority in the name of the profession on scientific questions, has often been advocated by THE PRACTITIONER. We therefore hail with satisfaction the amalgamation, partial as it is, that has been effected. It is not indeed the organisation of which we have dreamed, but it may perhaps be looked upon as an intermediate stage in its development. It is to be presumed that the financial difficulties, which at one time threatened to wreck the scheme, have been effectually overcome, and it may be hoped that the societies which still stand outside may before long see their way to cast in their lot with the new body. While rejoicing that the first step to a union of our scientific forces has been taken, we cannot help regretting that it has been decided to call the new body the Royal Society of Medicine. It is almost a colourable imitation of the title of the greatest and most ancient of our scientific associations, and it is to be feared that it will lend itself to misapprehension and even misuse. The public cannot be expected to discriminate very exactly between one Royal Society and another, and unfortunately it is not altogether inconceivable that some members might not take the trouble to dispel the confusion. We do not know whether the Royal Society will think it worth while to oppose on this ground the Charter that is to be asked for, but it would not be surprising if it did. Another objection to the name that has been chosen for the new body is that it will strengthen the hands of the not uninfluential section of the Fellows of the Royal Society which would say to doctors seeking admission within the sanctuary of the Temple of Science, *Procul, O procul este profani!* They will now be able to say, "You have a Royal Society of your own; that ought to be enough for you."

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The Strange Case of Mrs. Eddy.

In one of the most absurd of her many imaginings, Miss Marie Corelli described Pope Leo XIII. in his later years as a kind of living mummy, shown occasionally to the faithful, but, in a literal sense, having

> Neither wit, nor words, nor worth,
> Action, nor utterance, nor the power of speech,

and being, in fact, a galvanised corpse.

It may be that the newspaper scribes, who not long ago described Mrs. Eddy as "a rouged skeleton in the last stage of mental and physical decrepitude, who tottered painfully, and uttered a few incoherent words," showed a like power of invention. Her disciples on the other hand tells us that in conversation she displays great vigour of mind. It may be doubted whether the devotee of Christian Science can be accepted as a satisfactory judge of mental vigour. The son of the Prophetess is said to have reported that "she is weak in body, her mind is beclouded, and she is possessed by strange and irrational notions." There is a touch of pathos about this late discovery of a fact which has been patent to all intelligent readers of the writings that go by the name of Mrs. Eddy. These show on every page signs of a beclouded mind possessed by strange and irrational notions. Yet such as they are, the writings are said not to be her own, and Mark Twain in particular thinks her incapable of having produced them. Short of a diagnosis of downright imbecility, it would be impossible to express a meaner opinion of a human intellect than this.

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Mark Twain on Christian Science.

Mark Twain is known to all men as a humourist, but he can be very much in earnest when he likes. And he is very much in earnest in his handling of Christian Science and its Prophetess. His views were substantially given to the world in the *North American Review* two or three years ago, and they have now been published in fuller detail in a book which has lately appeared. He deals with it mainly as a system of religion, but as a large part of its strength as a religion lies in the power of healing that is claimed for it, much of his criticism is necessarily concerned with the aspect of the cult that specially interests the medical profession. He points out that, unlike other religions, Christian Science offers, in addition to heaven hereafter, health and a cheerful spirit in the present. "Who," he asks, "are attracted by Christian Science?" and his reply expresses the boundless scope as well as the strength of the appeal which it makes to the brain of "this foolish-compounded clay man." It appeals to those who are ailing in body or mind, and to those who have friends that are ailing in body or mind. "To mass it in a phrase, its clientage is the HUMAN RACE." It offers to rid the race of pain and disease. Can it do this? Mark Twain thinks that, in large measure, it can. He says:—

"How much of the pain and disease in the world is created by the imaginations of the sufferers, and then kept alive by those same imaginations? Four-fifths? Not anything short of that, I should think. Can Christian Science banish that four-fifths? I think so. Can any other (organized) force do it? None that I know of." The world would doubtless be a much pleasanter place if this were accomplished—both for the people who are well and for those fussy and fretting sick ones. Mark Twain thinks that in the meantime the Christian Scientist would kill off a good many patients, but probably not "more than get killed off now by the legalised methods."

He takes from the *Christian Science Journal* the following picture of the "average orthodox Christian," which, he says, is a true picture of the average human being:—

"He is a worried, and fretted, and fearful man; afraid of himself and his propensities, afraid of colds and fevers, afraid of treading on serpents or drinking deadly things."

Look on that picture and on this:—

"The average Christian Scientist has put all anxiety and fretting under his feet. He does have a victory over fear and care that is not achieved by the average orthodox Christian."

In the journal referred to "many of the redeemed testify and give thanks, and not coldly but with passionate gratitude. As a rule, they seem drunk with health, and with the surprise of it, the wonder of it, the unspeakable glory and splendour of it, after a long sober spell spent in inventing imaginary diseases and concreting them with doctor-stuff."

Mark Twain cites the testimony of several sufferers whose "claims" (the Christian Science jargon for the imaginary disturbance which "claims" to be an ailment, but has no real existence) have vanished, like ghosts at daybreak, before the incantations of Christian Science. These testimonies are accepted by Mark Twain without much demur, but an account of the restoration to perfect health, in a single night, of a fatally injured horse by Christian Science, makes him say, "I can stand a good deal, but I recognise that the ice is getting thin here." And after thinking the matter over, he comes to the sound conclusion: "Let us draw the line at horses. Horses and furniture."

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The True Inwardness of Christian Science

MARK TWAIN, we think, greatly exaggerates the sphere of influence of Christian Science; but he has a clear insight into the true inwardness of the cult. He says the power which the imagination has over the human body to heal it or make it sick is a force which is innate in man. "If left to himself, a man is most likely to use only the mischievous half of the force—the half which invents imaginary ailments for him, and cultivates them; and if he is one of these very wise people, he is quite likely to scoff at the beneficent half of the force, and deny its existence. And so, to heal or help that man, *two* imaginations are required: his own and some outsider's. The outsider B. must imagine that *his* incantations are the healing power that are curing A., and A. must imagine that this is so. I think it is not so at all; but no matter, the cure is effected, and that is the main thing." The outsider's work, he holds, is unquestionably valuable; he likens it to the work performed by the engineer when he handles the throttle and turns on the steam. "The actual power is lodged exclusively in the engine, but if the engine were left alone, it would never start of itself." Whether the engineer calls himself Christian Scientist, or Mind Curist, or Hypnotist, matters nothing. He is merely the engineer; he simply turns on the same old steam, and the engine does the whole work.

"In the case of the cure-engine it is a distinct advantage to clothe the engineer in religious overalls and give him a pious name. It greatly enlarges the business and does no one any harm." As to this we think Mark Twain has spoken in his haste. The cases that in the last few years have come before the law courts in this country have shown that Christian

Science is very far from harmless when it undertakes the treatment of real disease. But he has correctly explained its mode of action in that immense shadowland of disease which for lack of a better name we call "neurotic." The healing power of the mind has always been there, but Christian Science has organised that vast and wandering force and set it to work, and backed the business with capital, and concentrated its management in the hands of a small and very competent Trust. The results are seen, not in any diminution of the death rate, not in the abolition of infectious disease, not in the banishment of cancer and consumption from this vale where they cause so many tears, but in the increasing spread of a church run on the most strictly business principles. Mark Twain thinks it likely that the Christian Science Church will in 1910 have 10,000,000 adherents in America and 3,000 000 in Great Britain, and double those numbers in 1920, with an income approaching to £1,000,000 a day, and no expenses to be taken out of it, no taxes to pay, and no charities to support. It is remarkable that this religion which professes to cure all earthly ills, contributes nothing at all to any charitable object. Its mission is not to the Gentiles, but to the *Genteels*.

Scientific medicine, however, has nothing to fear from Christian Science.

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Old Quack Advertisements.

Philosophers have had much difficulty in providing a satisfactory definition of man. Plato's "biped without feathers" failed to stand a practical test. Man has also been defined as "a cooking animal," as being the only member of animated nature who cooks his food. But now the hungry phagocyte is supposed to have its diet daintily opsonized for it, cooking can no longer be held to be the *differentia proxima* between man and the lower animals. There is room, therefore, for a new definition of man, and we venture to propose "an animal deluded by advertisement" as meeting the requirements of the case. Man changes in everything else; he has rid himself of many superstitions, but he is still as much the prey of the advertiser as he was when he allowed himself to be gulled by the records of cures inscribed on the walls of the temples of Asklepios in ancient Greece. Many of these are exactly in the style of the infallible nostrums which are pillars of support for our free and independent press at the present day. And wherever we strike the trail of the advertiser through the ages, it is always the same kind of thing. Some chapters of Pliny's Natural History are simply quack advertisements. Paracelsus was an advertising quack with a considerable element of genius. Specimens of the advertisements of the Chevalier Taylor and other quacks of the eighteenth century have been given in recent numbers of THE PRACTITIONER. In looking over a reprint of the first number, dated December 4, 1791, of our highly respectable contemporary, the *Observer*, we find its pages swarming with quack advertisements. The virtues of "Dr. Howell's Celebrated Powders, the only effectual Remedy ever yet discovered for curing all kinds of Fits, particularly Epileptic or Falling Fits," are attested by grateful patients who had suffered many things from many physicians without benefit. Mr. Charles Angbaud, surgeon, described as "Grandson to the inventor, who was an eminent Apothecary to King William and Queen Anne," offers for sale "at his house, No. 78, the Angel and Trumpet, the upper end of St. Martin's Lane, Westminster," the Pectoral Lozenges of Blois, "so universally and satisfactorily known for their excellent properties in curing the Usual Diseases of the Lungs, as Colds, Tickling Coughs, Shortness of Breath (which they also contribute to sweeten), Asthmatic, Phthisicky and Consumptive Complaints." Mr. Smith, No. 5 (the corner of Golden Square), Lower John Street, gives the names of a few of his patients, who have been cured by his medicine of gout, rheumatism, and scurvy. Mr. Swainson, the sole proprietor of the genuine syrup of De Velnos, warns the public against counterfeits, adding:—"If, after repeatedly publishing the affidavits of Mr. and Madame De Velnos, and exhibiting the deeds which secure the secret of Mr. Swainson, weak and silly persons will believe the bare assertions of unprincipled impostors, they must suffer for their credulity." The whole philosophy of quackery is in these words. The *Gentleman's Magazine* for 1748 gives a list of the quacks then preying on the people of London, and their nostrums. It is interesting, and we think would amuse our readers, but want of space prevents our dealing with it here.

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Practical Notes.

The Cerebellum.—Each side of the cerebellum has three peduncles, the superior peduncle connects it chiefly with the opposite cerebral hemisphere, and partly with the cerebral hemisphere of the same side. The middle peduncle contains fibres, which connect the two halves of the cerebellum together in a physiological, though not in an anatomical, sense. The inferior peduncle connects the cerebellum mainly with the same side of the spinal cord. The upper end of the inferior cerebellar peduncle terminates in the vermis; in many of the lower animals, however, the vermis is practically the only part of the cerebellum which is present, and it is this portion of the cerebellum which is principally connected with the co-ordination of the movements of the body. If the cerebellum is removed in an animal, or if it is the seat of a cerebellar tumour in man, the result is some slight muscular weakness, more particularly on the same side of the body as that part of the cerebellum which is mostly affected. The chief symptom, however, is an inco-ordination of muscular movements, as is evidenced by a staggering gait, which is known as *cerebellar ataxy.* In order that the cerebellum may perform its functions in presiding over equilibration, it is necessary that it should receive impulses from without in order to keep it informed of the position of the body in space. These afferent impulses to the cerebellum are as follow:—(1) Kinæsthetic impressions travel to the cerebellum from the extremities, originating there in the skin, muscles, tendons, ligaments, and synovial membranes of the joints; these reach the cerebellum through the inferior cerebellar peduncle. Kinæsthetic impressions from the muscles of the eyes also reach the cerebellum through the superior cerebellar peduncle. (2) Visual impressions, arising, of course, in the eyes, reach the cerebellum by means of the superior cerebellar peduncle, they come through the optic thalamus. (3) Labyrinthine impressions arise in the cristæ acousticæ of the ampullæ of the three semi-circular canals, and in the maculæ acousticæ of the utricle and saccule of the internal ear, and travel by the auditory nerve to the hind brain, thence by the middle and inferior cerebellar peduncles to the cerebellum. The cerebellum then sends out impulses, by means of the fibres, which arise in the cells of Purkinje, chiefly to the opposite half of the cerebral cortex, but some go to the cerebral cortex of the same side. It also sends out impulses to influence the nuclei of the cranial nerves situated in the mid- and hind-brain, and, lastly, impulses which travel down the spinal cord. In this way, the cerebellum influences the discharge of the nervous impulses from the cells of the cerebral cortex, from the nuclei of the cranial nerves, and from the large multipolar nerve cells which are situated in the anterior cornua of the spinal cord. In this way the cerebellum presides over the co-ordinate movements of the muscles of the body.

CEREBELLAR TUMOURS.—In the majority of the cases of neoplasms of the cerebellum, the following symptoms are usually present :—1. *Headache*, which varies considerably in intensity, and is usually referred to the back of the head, where there may be an area of pain which is increased on deep pressure. 2. *Nausea and vomiting*, which occur without any relation to food. 3. *Optic neuritis*, which is more constant in cerebellar tumours than in any other intracranial tumours. The development of the optic neuritis, or choked disc, is often very rapid ; choking of the disc is usually extreme, and hæmorrhages in the retina are numerous, especially in the later stages of the disease. 4. *Vertigo*, which Dr. Charles K. Mills, of Philadelphia, explains as follows :—The vertigo, which is so frequently a general symptom of brain tumour, wherever situated, is usually due to irritation of branches of the trigeminal nerve, which are distributed near the inner surface of the dura, the irritation of the fifth nerve being reflected to the bulbar nuclei of this nerve, and thence to the pneumogastric nucleus. This is usually one of the causes of the vertigo in tumours of the cerebellum, when the growth is connected with the dura, which, however, is not the rule. In other cases, it should be regarded as a focal rather than as a general symptom of cerebellar tumour, as it is caused by the disturbing influence exerted by the tumour upon the cerebello-vestibular apparatus. 5. *Nystagmus ;* the movements are sometimes rapid and fine, sometimes slow and coarse. 6. *Muscular weakness*, which is a real cerebellar symptom.

TÂCHE CÉRÉBRALE.—If the finger-nail is gently drawn across the skin of a normal individual, a fine white line is momentarily produced, which may be followed by the appearance of a slight active flush in the trail of the finger-nail, this flush then very soon disappears. The appearance of the white line, so produced, is due to the local anæmia resulting from the pressure, and is soon followed by hyperæmia; the *tone* of the capillaries is then rapidly recovered, and the portions of the skin, which have been stimulated by pressure of the nail, soon regain their normal appearance.

In some of the toxæmic conditions, which occur in patients suffering from certain infective diseases, the *tâche cérébrale* is readily produced. If the finger-nail is drawn across the skin of the patient's face, abdomen, arm, or thigh, the white streak mentioned above is rapidly followed by a bright red streak, which usually lasts much longer, is of a deeper colour, and is considerably broader than it is in healthy persons. In the second stage of *tuberculous basal meningitis*, when the irritative symptoms have subsided, and vomiting is no longer a marked symptom, and the child has passed into a dull apathetic condition, the head remains retracted, and the child utters an occasional cry, the tâche cérébrale can then be readily obtained, though it has no particular diagnostic significance. In persons suffering from *enteric fever*, the tâche cérébrale may be readily produced, but, as Professor Osler points out, it is a vaso-motor phenomenon of no special significance. In some cases of *epidemic cerebro-spinal meningitis*, the tâche cérébrale may be easily obtained, but because of the facts mentioned above, too much importance must not be attached to its production, and other symptoms, such as the sudden onset of the disease, the severe headache, the pain in the back and in the limbs, the elevation of temperature, the rigidity of the muscles of the neck and back, and the presence of Kernig's sign must be examined for. In order that the diagnosis may be completed, a lumbar puncture must be performed, and the cerebro-spinal fluid, so obtained, examined for the diplococcus intracellularis meningitidis, which was described by Weichselbaum in 1887; in the tissues, this organism is almost constantly found within the polynuclear leucocytes.

NOCTURNAL ENURESIS.—The most frequent causes of this bad habit are hyperacidity of the urine and abnormally deep sleep. When the former is proved to be present, it is usually enough to give alkaline remedies in order to obtain a cure. When the wetting occurs as a consequence of too deep a sleep, the best measure to adopt is systematic awakening of the child. In order to carry out this method thoroughly in the case of inveteracy, and to force the child to get accustomed to waking up at the critical moment, the following procedure has been tried with success:—The child lies upon a mattress composed of two layers of metallic tissue separated by a dry cloth (woollen or flannel). Each of these layers is connected by a wire to the working part of a loud electric bell placed above the bed. As soon as the first drop of urine has damped the cloth, and so made contact, the bell rings furiously, and wakens the sleeper. By reflex inhibition, the action of the bladder is stayed. From two to five repetitions of this experience are said to be sufficient for a radical cure. (*Gazette Médicale de Paris.*)

Before the above-suggested treatment is carried out, all possible sources of reflex irritation must be carefully eliminated, such as threadworms in the rectum, stone in the bladder, an elongated, contracted, or adherent prepuce. Relief has been known to follow the removal of adenoids. A good plain, non-stimulating diet is best for the child, and no evening meals should be taken. The child should be taught to sleep on either side, not on the back, and, as suggested above, awakened at certain intervals. Attention to these matters often brings about a cure, but should they fail then drug treatment may be resorted to. Belladonna is the best remedy, and children bear it well. Sir William Whitla suggests the following mixture to be prescribed for a child 7 years old:—

℞. Potassii Bromidi - - - ʒv.
Tincturæ Belladonnæ - - - ʒiv.
Syrupi Simplicis - - - ℥i.
Aq. Floris Aurantii - - ad ℥iv.

Misce. Fiat mistura, cujus capiat cochleare unum minimum vespere et hora somni.

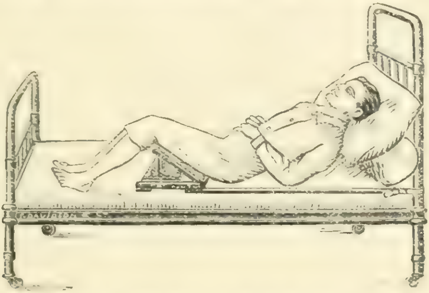
It is found that Liquoris Strychninæ Hydrochloridi, combined with Liquoris Atropiæ Sulphatis, has given excellent results.

Preparations, Inventions, etc.

HIP ELEVATOR OR NON-SLIPPING BED REST.

(Messrs. F. Davidson & Co., 29, Great Portland Street, W.)

This bed rest is designed to prevent a patient from slipping down in bed. It is ingeniously constructed so as to be fitted at any convenient angle, and, by means of straps, can be made to remain at any desired position on the mattress.



By placing the rest in the reverse way under the shoulders of the patient, it can manifestly be used as an elevator for the head and shoulders. The framework is of oak, and the fitting of special flexible spring wire. The many conditions under which the patient will derive comfort and advantage by the use of this bed rest are obvious, and we have no hesitation in heartily recommending it to the profession.

THE "GLASEPTIC" NEBULISERS AND SPRAYS.

(Messrs. Parke, Davis & Co., 111, Queen Victoria Street, E.C.)

These ingenious instruments are exceedingly simple in construction, and, being practically in one piece, are very easily manipulated. Apart from the india-rubber bellows and tubing, the construction is entirely of glass, hence the liability of oxidation and corrosion is avoided. They are readily cleansed, and can consequently be made perfectly aseptic, and, from the peculiarity of their construction, they can be used with a very small quantity of liquid. The *Nebuliser* produces an extremely fine nebula with solutions having an oily base, and is also effective for nebulising solutions of whatever density or viscosity, whether spirituous, oily, or aqueous, and for concentrated saline solutions, or viscid preparations such as Adrenalin Inhalant. For nasal use the neck of the apparatus is inserted into the nostril, while, for the treatment of the pharyngeal or laryngeal regions, a throat piece is fitted on to the neck and the flattened end placed between the lips. The *Spray* produces a spray of a less finely divided character than the nebula, and, although primarily intended for spraying aqueous or alcoholic solutions, may be used with oils of a light specific gravity. Both are made of conveniently small dimensions and are exceedingly compact, so that, if required, they may be carried in the pocket, or in a small bag.

NEW "OKOL" AND "SANITAS FLUID."

(Sanitas Company Limited, Locksley Street, Limehouse, E.)

New "*Okol*" is a disinfectant just introduced by the Sanitas Company, and has been satisfactorily reported on by Mr. M. Wynter Blyth. His report shows that the tests were carried out with a 24 hours' old culture of B. Typhosus in nutrient broth (−1·5) at a temperature of 17·5 C. As compared with carbolic acid, the germicidal value of Okol was found to be 22, thus constituting the new fluid one of the most powerful and efficacious disinfectants on the market.

"*Sanitas Fluid*" is exceedingly useful as a spray for purifying the vitiated air of dwelling-rooms, and especially rooms in which disease germs are existent. The necessity of keeping the air of sick rooms perfectly sweet and pure is now fully and completely recognised, and, for this purpose, the use of "Sanitas Fluid" is valuable. It is well to remember, too, that mixed with water, in the proportion of 1 to 4, it forms an excellent mouth wash, for, by its use, harmful bacteria are destroyed, and the teeth and mouth kept perfectly aseptic.

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LLANDUDNO—NURSING HOME.

(Miss Bennetts, Mostyn Avenue, Llandudno.)

During the last thirty years Llandudno has developed, from an unpretending little seaside town on the north coast of Wales, into a very popular health resort, and this popularity is due to its position, its equable temperature, and the purity and dryness of its atmosphere. The advantages of a well-ordered nursing home in such a peculiarly healthy spot are paramount, and members of the profession will doubtless be glad to have knowledge of such an establishment in Llandudno.

THE PRACTITIONER.

MAY, 1907.

FURTHER OBSERVATIONS ON HEART-BLOCK.

By G. A. GIBSON, M.D., F.R.C.P. (Edin.),
Physician to the Royal Infirmary, Edinburgh; and

W. T. RITCHIE, M.D., F.R.C.P. (Edin.),
Clinical Assistant Pathologist to the Royal Infirmary, Edinburgh.

By clinical observation and experimental investigation, it has now been conclusively proved that disturbances of both the rate and rhythm of the heart may be the manifestation of impairment of its conductivity. In the heart of the tortoise and of the frog, we know that the stimuli to contraction, which normally originate in the sinus venosus, and pass thence to the auricular and the ventricular fibres, may be blocked at various points, but, in particular, at the junction of the sinus and auricle, and again at the auriculo-ventricular boundary. Defective conduction of a stimulus may be held as proved, whenever the period, elapsing between the contraction of the sinus and the auricle, or of the auricle and the ventricle, is unduly lengthened.

The study of the conductivity of the mammalian heart has, however, been almost entirely restricted to that of the fibres of the auriculo-ventricular bundle, which constitute the functional bond of union between the auricles and the ventricles. That there must be a muscular connection between the auricles and the ventricles has been stoutly maintained ever since Gaskell[1] recorded his observations on the heart of the tortoise, but although our knowledge of the site, distribution, and structure of the connecting bundle—the auriculo-ventricular bundle—was advanced by the observations of Stanley Kent,[2] His junior,[3] Retzer,[4] and Braeunig,[5] it is only by the recent observations of Tawara,[6] which have been

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[1] *Journal of Physiology*, 1883, Vol. IV., p. 43.

[2] *Journal of Physiology*, 1893, Vol. XIV., p. 233.

[3] *Arbeiten aus der med. Klinik zu Leipzig*, 1893, S. 14–49; *Wiener med. Blätter*, 1894, XVII., S. 653; *Centralbl. f. Physiol.*, 1896, Bd. IX., S. 469.

[4] *Archiv f. Anatomie u. Physiologie*, Anatomische Abteilung, 1904, S. 1.

[5] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1904, Supplement-Band, S. 1.

[6] "*Das Reizleitungssystem des Saugetierherzens*," 1906.

verified by Keith and Flack,[1] that an accurate conception of its arrangement and distribution has been formed. Tawara has shown that, within the auricular walls, there exists a network of special muscle fibres with a nodal point in the inter-auricular septum just above the attachment of the mesial segment of the tricuspid valve. From the nodal point, a bundle of muscle fibres passes downwards through the fibrous auriculo-ventricular ring, to divide in the inter-ventricular septum into two branches, one for each ventricle. The fibres of each branch subsequently pass as Purkinje's fibres in the subendothelial tissue, and ultimately terminate in the ordinary cardiac muscle fibres in the walls of the ventricles, and in their papillary muscles. Those, who uphold the myogenetic theory of the rhythmic action of the heart, believe that the fibres of the auriculo-ventricular bundle are mainly concerned with the conduction of stimuli to contraction, but it should not be forgotten that the truth of the myogenetic theory has recently been called in question by Hering,[2] and also by Carlson,[3] who has made a most careful study of the action of the heart in *Limulus*. It is admitted, on all hands, that the auriculo-ventricular bundle contains not only nerve fibres but ganglia, as Tawara[4] has clearly shown. That the conduction is by muscle is, as yet, therefore unproved.

After section of the main stem of the auriculo-ventricular bundle, as performed experimentally by His junior,[5] Humblet,[6] and Hering,[7] no stimuli to contraction can be transmitted onwards from the auricles to the ventricles, nor can retrograde extra-systoles pass from the ventricles to the auricles, and while the rhythm and rate of the auricular contractions continue unchanged, the ventricles beat less frequently than the auricles, and with independent rhythm. Section of the bundle, therefore, results in complete dissociation of the auriculo-ventricular rhythm—complete heart-block.

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[1] *Lancet*, 1906, I., p. 623; *Ibid.*, 1906, II., p. 359.

[2] *Zeitschrift fur experimentelle Pathologie und Therapie*, 1906, Band III., S. 511.

[3] *American Journal of Physiology*, 1905-06, Vol XV., p. 113.

[4] *Zentralblatt für Physiologie*, 1906, Bd. XIX., S. 300.

[5] *Wiener med. Blätter*, 1894, Bd. XVII., S. 653: *Centralbl. f. Physiologie*, 1896, Bd. IX., S. 469.

[6] *Arch. intern. de Physiologie*, 1906, tome III., p. 330.

[7] *Archiv f. d. gesammte Physiologie*, 1905, Bd. CVIII., S. 267; *Ibid.*, 1906, Bd. CXI., S. 298.

The results of experimental clamping of the auriculo-ventricular bundle have been studied by Erlanger,[1] by that observer and Hirschfelder,[2] and by von Tabora.[3] By gradually increasing the pressure of the clamp, the conductivity of the fibres of the bundle can be gradually lessened, so that only every alternate, and, subsequently, only every third, fourth, or fifth stimulus is transmitted onwards from the auricle to the ventricles. Thus the ratio between the auricular contractions and the ventricular may be 2 : 1, 3 : 1, 4 : 1, etc., and the condition, which then prevails, is one of partial heart-block. When the pressure of the clamp is still further increased, no stimuli can pass from the auricles to the ventricles, the block which was previously partial becomes complete, and there is dissociation of the auriculo-ventricular rhythm.

It is now well established that both partial and complete block at the auriculo-ventricular bundle may be met with in the human heart. In former papers [4,5] we have recorded such cases, which we studied by graphic methods, the fluorescent screen, and the capillary electrometer, and we have described the effect of atropine administration upon the rate of the auricles and of the ventricles in complete auriculo-ventricular heart-block. Within the last two years, the subject of auriculo-ventricular heart-block has engaged the attention of many other observers, and tracings demonstrating that condition have been recorded by Mackenzie,[6] Rihl,[7] Belski,[8] Hay,[9] Bönninger,[10] Erlanger,[11] Brouardel and Villaret,[12] Leuchtweis,[13] Finkelnburg,[14]

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[1] *Journal of Experimental Medicine*, 1906, Vol. VIII., p. 8.

[2] *American Journal of Physiology*, 1906, Vol. XIV., p. 153.

[3] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III., S. 499.

[4] "*The Nervous Affections of the Heart*," 1904, p 61; *Edinburgh Medical Journal*, 1905, Vol. XVIII., New Series, p. 9; *British Medical Journal*, 1906, II., p. 22; *Ibid.*, 1906, II., p. 1113.

[5] *Proceedings of the Royal Society of Edinburgh*, 1905, Vol. XXV., p. 1085.

[6] *British Medical Journal*, 1905, I., p. 521; *Ibid.*, 1906, II., p. 1109.

[7] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1905, Bd. II., S. 83.

[8] *Zeitschrift f. klinische Medizin*, 1905, Bd. LVII., S. 529.

[9] *British Medical Journal*, 1905, II., p. 1034; *Lancet*, 1906, I., p. 139; *Medical Chronicle*, 1906, Vol. 44, p. 371.

[10] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1905, Bd. I., S. 663.

[11] *Journal of Experimental Medicine*, 1905, Vol. VII., p. 676.

[12] *Archives de médecine expérimentale et d'anatomie pathologique*, 1906, tome XVIII., p. 230.

[13] *Deutsches Archiv f. klinische Medicin*, 1906, Bd., LXXXVI., S. 456.

[14] *Ibid.*, 1905, Bd. LXXXII., S. 586; 1906, Bd. LXXXVI., S. 462.

Magee Finny,[1] Roos,[2] Hay and Moore,[3] Bard,[4] Lichtheim,[5] Wenckebach,[6] Goteling Vinnis,[7] Schmoll,[8] Joachim,[9] and Ashton, Norris, and Lavenson.[10] The case recorded, in 1900, by Votruba[11] was not referred to in our previous papers, but may now be accepted as a heart-block, whereas the exact nature of the cases recorded by Perugia[12] and Livierato[13] must remain somewhat uncertain in the absence of graphic records of the jugular pulsations and ventricular contractions. The only other method, whereby heart-block has been graphically demonstrated, is that recently described by Einthoven,[14] who shows that, by the method which he has devised for graphically recording the electro-motive changes of the heart, a dissociation of the auriculo-ventricular rhythm can be as accurately studied by the electro-cardiogram as by tracings of the jugulo-carotid pulsations.

Although a lesion of the auriculo-ventricular bundle has been experimentally shown to produce heart-block, and, although morbid changes have been described in that bundle of the human heart by Stengel,[15] one of us,[16] Schmoll,[17] Jellick, Cooper, and Ophuls,[18] and Keith and Miller,[19] it must be borne in mind that a partial, or possibly even a complete block at the auriculo-ventricular bundle may be induced by vagus stimulation, as was observed by Knoll[1] after the administration of helleborein, and by Cushny[2] and Mackenzie[3] after the employment of digitalin. By means of calycanthine, Cushny[4] has also been able to cause heart-block in a pithed frog, but, in this experiment, the block was almost certainly due to a direct action upon the heart muscle, the inhibitory mechanism not being involved. Von Tabora[5] has recently investigated, by experimental methods, the auriculo-ventricular block induced by digitalin, and has shown that it is due partly to the effect of the drug on the vagus, and also to a direct effect upon the auriculo-ventricular bundle. When the bundle is intact, digitalis may of itself undoubtedly cause a partial block at that point, but probably never a complete auriculo-ventricular dissociation, whereas, when the conductivity of the fibres of the bundle is already impaired by structural change, the administration of digitalis may convert a pre-existing partial block into one that is complete. That stimulation of the vagus has an effect upon the conductivity of the fibres of the auriculo-ventricular bundle has also been proved by Mackenzie[6] in a patient in whom, although there was no pre-existing block, the conductivity of the heart was impaired. In that patient, the act of deglutition induced a transient block at the auriculo-ventricular bundle.

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[1] *British Medical Journal*, 1906, I., p. 967; *Transactions of the Royal Academy of Medicine in Ireland*, 1906, Vol. XXIV., p. 99.

[2] *Zeitschrift f. klinische Medizin*, 1906, Bd. LIX., S. 197.

[3] *Lancet*, 1906, II., p. 1271.

[4] *Journal de Physiologie et de Pathologie général*, 1906, tome VIII., p. 466, and p. 473.

[5] *Deutsches Archiv f. klinische Medicin*, 1905, Bd. LXXXV., S. 360.

[6] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1906, S. 297.

[7] De aanhoudende verdubbeling van den hartslag (hart-bigeminie), Leiden. *Proefschrift*, 1905.

[8] *Deutsches Archiv f. klinische Medicin*, 1905, Bd. LXXXVII., S. 554.

[9] *Ibid.*, 1906, Bd. LXXXV., S. 373; 1907, Bd., LXXXVIII., S. 574.

[10] *American Journal of the Medical Sciences*, 1907, Vol. CXXXIII., p. 28.

[11] *Sbor Fraci*, 1900, p. 123.

[12] *Gazetta degli Ospedale e delle Cliniche*, 1905, XXVI., p. 859.

[13] *La Clinica Medica Italiana*, 1906, XLV., p. 71.

[14] *Archives Néerlandaises des Sciences exactes et naturelles*, Série II., tome XI., 1906, p. 239.

[15] *American Journal of the Medical Sciences*, 1905, Vol. CXXX., New Series, p. 1083.

[16] *British Medical Jonrnal*, 1906, II., p. 1118.

[17] *Deutsches Archiv f. klinische Medicin*, 1906, Bd. LXXXVII., S. 554.

[18] *Journal of the American Medical Association*, 1906, Vol. XLVI., p. 955.

[19] *Lancet*, 1906, II., p. 1429.

Whereas the conductivity of the mammalian heart, as we have already stated, has been studied almost exclusively at the auriculo-ventricular bundle, we are now able to record a case in which there was a block at that bundle, together with a marked depression of conductivity at the junction of the superior vena cava with the right auricle.

A cabman, *æt.* 66, came under our observation in July 1906, complaining of weakness and shortness of breath. For thirty years, he has indulged in occasional drinking-bouts, but has taken almost no alcohol during the last eight years. He acquired syphilis nine years ago; treatment was not continued for more than three weeks. Six years ago, he suffered from a severe attack of acute rheumatism, and, for the last six years, he has suffered from chronic bronchitis, being often confined to bed for 6–8 weeks during the winter time. He gives a history of occasional syncopal attacks. Their onset is sudden, although they are ushered in by a choking sensation in the throat, and "and a terrible buzzing noise in the ears." During an attack, he has, on several occasions, dropped on to the floor or on the street, the period of unconsciousness lasting from a few seconds to a couple of minutes. It does not appear that there were ever any epileptiform convulsions during an attack.

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[1] *Sitzungsberichte d. k. Akad. d. Wiss. in Wien*, 1894, Bd. CIII., Abt iii. (Math. Naturioiess. Classe), S. 298.

[2] *Journal of Experimental Medicine*, 1897, Vol. II., p. 232.

[3] *British Medical Journal*, 1905, I., p. 587.

[4] *Archives internationales de Pharmacodynamie et de Therapie*, 1905, tome XV., p. 487.

[5] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III, S. 499.

[6] *British Medical Journal*, 1906, II., p. 1109.

The patient is not cyanosed or dropsical. The cardiac impulse, which is visible only in the 5th left interspace, 1 inch external to the left mammary line, and 5 inches from the mid-sternal line, is somewhat feeble, but rhythmical; the rate when the patient was first seen was 35 per minute. The borders of the heart lie 2 inches to the right and 5 inches to the left of the mid-sternal line. On auscultation at the mitral area, there is a soft blowing systolic murmur, while a soft diastolic murmur follows the second sound, but no presystolic murmur is audible. At the other areas, both the first and second sounds are closed; the aortic second sound is accentuated. On auscultating over the inner end of the 2nd and 3rd left interspaces, one or more faint additional sounds are distinctly heard during the long interval elapsing between the second sound and the subsequent systolic sound. Those additional sounds are to be regarded as caused by auricular systoles. When the patient is examined with the fluorescent screen, the auricular pulsations are seen to be at least twice as numerous as those of the ventricles.

The radial arteries are thickened, but are not obviously tortuous. The rate of the pulse, when the patient first came under observation, was 35 per minute, and it was quite regular. The arterial blood pressure is high. Faint yet distinct pulsations are visible in the jugular veins at the root of the neck. Both these features will be considered in a later part of this paper.

The thorax is enlarged in its antero-posterior diameter,

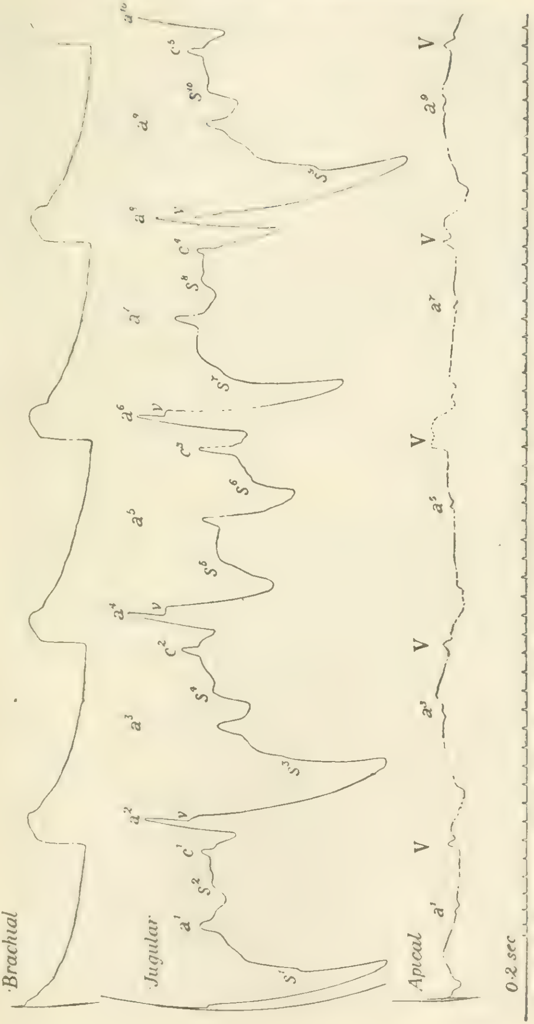

Fig. 1.—Partial auriculo-ventricular heart-block, and defective conductivity at the junction of the superior vena cava and right auricle.

and expanded feebly during deep inspiration. On percussion, the lungs are found to encroach upon the heart and the complementary pleural sinuses. The breath sounds are everywhere accompanied by sibilant rhonchi, and at the bases, posteriorly, by a few medium crepitations. There is much coughing, attended with an abundant yellowish muco-purulent expectoration. The urine contains a few granular casts, but is otherwise of normal character. The other systems of the body present no further particular features which need to be recorded.

Many tracings were taken from the patient. They proved that the block at the auriculo-ventricular bundle was at first partial (Figs. 1 and 2), but afterwards complete (Figs. 3, 4, 5, and 6). When we analyse the tracings, reproduced in Fig 1, we find, first, that there is distinct evidence of a partial heart-block as a result of defective conductivity at the auriculo-ventricular bundle. In the figure, there is reproduced a portion of three tracings taken simultaneously from the jugular vein, the brachial artery, and the apex-beat of the heart. The chronographic record indicates the time in fifths of seconds. In the tracing from the jugular vein, the normal sequence of the auricular, *a*, carotid, *c*, and ventricular, *v*, waves is disturbed, in that every auricular wave is not followed by a carotid wave, and that, when a carotid wave does follow an auricular wave, the *a—c* interval, instead of being equal to 0·2 of a second, as in health, is equal to either 0·7 or 0·8 of

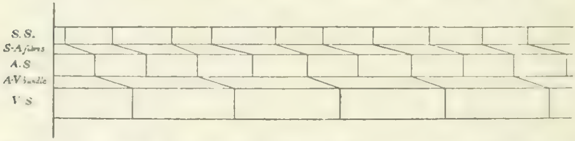

Fig. 2.—*Diagram constructed to illustrate the events recorded in Fig. 1. The vertical and oblique lines indicate the stimuli to contraction passing from the superior vena cava (S.S.) to the auricles (A.S.), and thence to the ventricles (V. S.). Each venous contraction evokes an auricular response, but there is defective conductivity at the fibres which connect the superior vena cava and the auricle (S-A fibres). At the A-V bundle, conductivity is so depressed that only every alternate stimulus is conducted from auricle to ventricle, so that there is partial heart-block.*

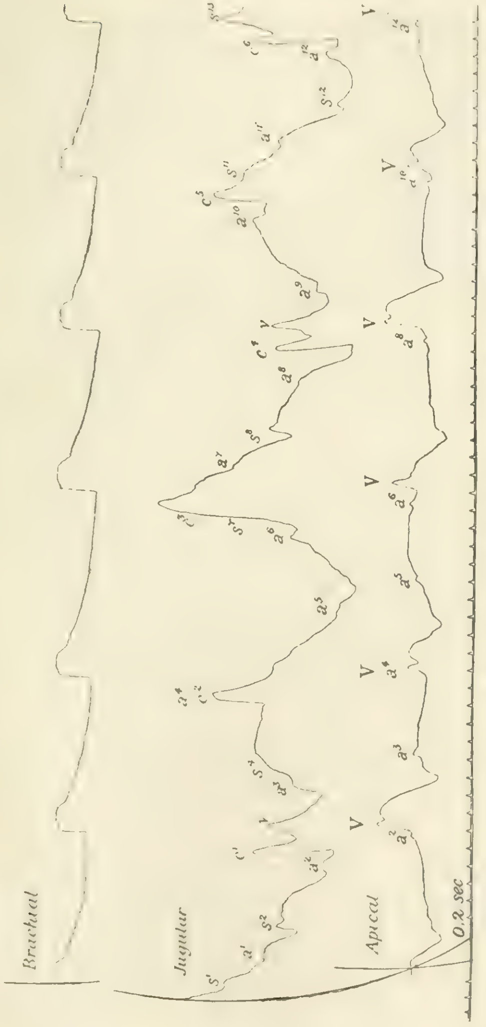

Fig. 3.—Complete auriculo-ventricular heart-block, and defective conductivity at the junction of the superior vena cava and right auricle.

a second. This lengthening of the *a—c* interval is due to the conductivity of the fibres of the auriculo-ventricular bundle being so depressed that the transmission of the stimuli from the auricles to the ventricles takes four times as long as in health ; and the impairment of conductivity is so pronounced that only every alternate stimulus can pass onwards to the ventricles, as is diagramatically represented in Fig. 2. In Fig. 1 the rate of the auricular contractions is 55·6 per minute, while that of the ventricular beats is 27·8 per minute.

When the tracings, reproduced in Fig. 3, were taken six days later, the block at the auriculo-ventricular bundle had become complete. The complete dissociation of the auricular rhythm from the ventricular is proved by comparing the rhythm of the auricles with that of the ventricles. The rate of the auricular contractions is 65·2 per minute, that of the ventricular beats is 32·4 per minute. In the jugular tracing, the wave *a* sometimes precedes the wave *c*, at others, it coincides with, or quickly follows after the latter, while, in the apical tracing, the waves *a* are seen to have a constantly changing time relationship to the ventricular systoles, *V*. If further proof of the complete dissociation of ventricular rhythm from the auricular were required, it would be furnished by the tracings which were taken at a later date, and of which a portion is reproduced in Fig. 5. There we find not only a

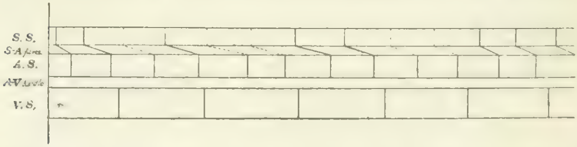

Fig. 4.—*Diagram to illustrate the events recorded in Fig. 3. The 1st, 2nd, 4th, 7th, 8th, 11th, 12th and 13th contractions (S. S.) of the superior vena cava, which were recognisable in the jugular tracing of Fig. 3, are indicated by continuous vertical lines ; the 3rd, 5th, 6th, 9th and 10th contractions of the vena cava could not be recognised in the tracing, and are therefore indicated by interrupted vertical lines. There is defective conductivity at the fibres connecting the superior vena cava and the right auricle. While the auricles beat rhythmically (A. S.), no stimuli to contraction are transmitted over the A-V bundle ; there is complete dissociation of the ventricular rhythm (V. S.) from the auricular.*

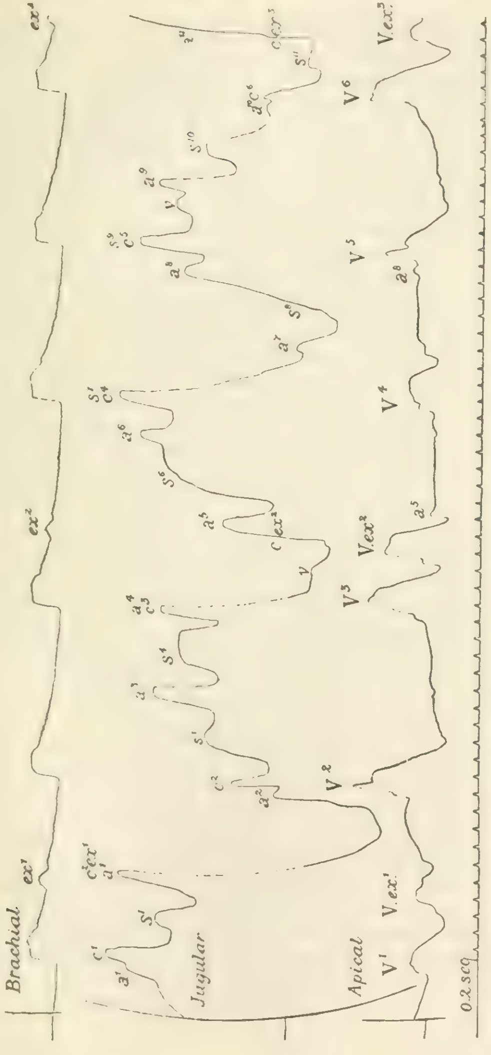

Fig. 5.—*Complete auriculo-ventricular heart-block, and also defective conductivity at the junction of the superior vena cava and right auricle, with ventricular extra-systoles.*

similar inconstancy in the time relation between the waves *a* and *c* in the jugular tracing, and the waves *a* and *V* in the apical tracing, but observe that the length of the post-extra-systolic pause (the compensatory pause) after the second extra-systole, $V\,ex^2$, is exactly equal to the subsequent inter-systolic period. This point is to be observed in the apical tracing; in the tracing from the brachial artery, the post-extra-systolic pause after the extra-systole, ex^2, is appreciably shorter than the inter-systolic period following it, because of the well-recognised delay in the transmission of the extra-systolic pulse wave into the artery. The first extra-systole, $V\,ex^1$, recorded in Fig. 5, is an interpolated one, for the period, that elapses between the commencement of V^1 and V^2, is exactly equal to that between the commencement of V^2 and V^3.

In order to determine whether the dissociation of the ventricular rhythm from the auricular was, in this case, due solely to structural changes in the auriculo-ventricular bundle, or was entirely, or partly, due to dromotropic influence of vagus origin, tracings were taken before and after the administration of $\frac{1}{50}$ gr. of atropine, hypodermically. Prior to the injection of this drug, the heart-block was complete, and the rate of ventricular systole equal to 34·3 per minute. Three minutes after $\frac{1}{50}$ gr. of atropine, the block had disappeared; each auricular systole evoked a ventricular response, the rate of contraction being 58·8 per minute. The conductivity of the auriculo-ventricular bundle was still, however, markedly lower than in

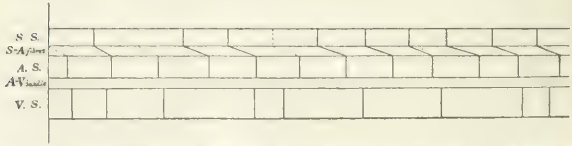

Fig. 6.—*Diagram to illustrate the events recorded in Fig. 5. The 2nd and 5th contractions of the superior vena cava (S. S.) are indicated by interrupted vertical lines. Each contraction of the vena cava is followed by an auricular contraction, although the conductivity of the fibres between the vein and the auricle is impaired. No stimuli to contraction pass over the A-V bundle to the ventricles. The ventricular contractions (V. S.) are irregular.*

health, for the *a—c* interval was equal to 0·6 of a second. Fifteen minutes after the administration of the drug, the block was still absent, the rate of contraction of the auricles and ventricles being 37·5 per minute; the *a—c* interval was only 0·4 of a second. In thirty minutes, there was again a partial block at the auriculo-ventricular junction; the ventricular rate was 34·7 per minute. One hour after the dose of atropine, the block was again complete, and the ventricular rate had fallen to 32·7 per minute. As the administration of atropine improved the conductivity of the fibres of the auriculo-ventricular bundle so markedly as to lead to the transient disappearance of the block at that bundle, we are justified in concluding that, in this patient, the heart-block was at least partly due to negative dromotropic influence of the vagus. But as the heart-block was known to have persisted for several months, and to have been complete, we are confident in asserting that, in addition, there must have been some structural change in the auriculo-ventricular bundle.

One of the most interesting points in the case, is the graphic record of a marked depression of conductivity in the fibres connecting the superior vena cava with the right auricle. Wenckebach[1] and Hering[2] are the only two observers who have as yet brought forward evidence to prove that there may be a block at that point in the mammalian heart comparable with a block at the sino-auricular junction of the heart of the tortoise, as recognised by Gaskell.[3] One series of tracings, which Wenckebach analyses, was obtained from a man thirty years of age. The disturbance of rhythm of the auricles and ventricles, in that case, justifies the conclusion that, while the rhythm of the fibres above the right auricle was regular, the auricular allorrhythmia was due to dropping of auricular beats consequent upon a partial block at the junction of the muscular fibres of the superior vena cava with those of the right auricle. In another case, Wenckebach shows that auricular systoles were dropped because of depression of excitability of the fibres between the vein and the auricle. A similar depression of excitability at the auriculo-ventricular fibres, leading to dropping of ventricular beats, has already been recorded by one of us,[1] as well as by Hay,[2] and by Wenckebach.[3]

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[1] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1906, S. 318.

[2] *Loc. cit.*

[3] *Journal of Physiology*, 1883, Vol. IV., p. 43.

In 1900, Hering[4] observed that, in rabbits, when the rate of the exposed heart decreased on cooling, the auricular pulsations were seen to distinctly follow those of the great veins, that, subsequently, an auricular contraction would occur only after several pulsations of the veins, and that, eventually, the latter pulsated alone without any accompanying auricular beats. The same observer has recently[5] obtained tracings from a rabbit and a dog, both presenting a similar disturbance of cardiac rhythm, which can only be explained by the assumption of a transient defect of conductivity of fibres between the site, at which the stimuli to contraction originate, and the auricles. These are the sole contributions, so far as we are aware, which have hitherto been made to either prove or disprove the possibility that affections of conductivity may be met with at the junction between the venæ cavæ and the right auricle of the mammalian heart.

In the jugular tracings of Figs. 1, 3, and 5, we find, in addition to the waves a, c, and v, yet another wave, s, more or less well marked throughout the tracings. In Fig. 1 the wave s precedes the auricular wave, a, by 0·6—0·8 of a second. The wave s is not an accidental one, for it is recognisable in all the tracings taken from the patient. It is not due to a ventricular extra-systole, for neither in the cardiogram nor in the sphygmogram is there evidence of such an event. Nor is the wave s due to an auricular systole, for whereas the auricular waves of the jugular tracing (*see* Fig. 1), with the exception of those which occur at a time when the ventricles are in systole, are also represented by distinct waves, a, in the cardiogram, the waves s are not represented in the latter. The wave s is sometimes followed immediately by an auricular wave, for example, s^{12} and a^{12} in Fig. 3, also s^3 and a^3 in Fig. 5. At other times, a carotid wave is interposed between them, as between s^1 and a^4, and between s^8 and a^8 in Fig. 1 ; and when there is complete dissociation of the rhythm of the auricles and of the ventricles, the wave *s* may coincide with the wave *v*, or with the carotid wave. The latter event is observed in the broad topped waves s^7 c^4, and s^9 c^5 in Fig. 5.

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[1] *British Medical Journal*, 1906, II., p. 1113.

[2] *Lancet*, 1906, I., p. 139.

[3] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 906, S. 328.

[4] *Archiv f. die gesammte Physiologie*, 1900, Bd. LXXXII., S. 21.

[5] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III., S. 511.

The wave *s* is, we believe, the expression of the rise in pressure in the jugular vein induced by the rhythmical contraction of the muscular fibres of the superior vena cava. That the venæ cavæ pulsate was known by Haller,[1] and Johannes Müller,[2] but the fact had been forgotten until Lauder Brunton, and Fayrer[3] again drew attention to the rhythmical contractile power of the pulmonary veins and of the vena cava.

The possibility that the contraction of the muscular fibres of the superior vena cava may give rise to an appreciable wave in the jugular vein is admitted by Erlanger,[4] who says that the auricular systole may be preceded by a distinct positive wave in the veins of the neck. In normal circumstances, this wave cannot be observed, but Erlanger believes it is possible that, when the pressure in the great veins is high, the effect of their contraction may become evident. Wenckebach[5] also admits that such a wave may be recognisable in tracings. He says, "I cannot but believe that the difficulty so frequently experienced in determining precisely the commencement of the *a* wave is due to a slight elevation preceding it." In one of his cases, Wenckebach draws attention to the fact that the auricular waves were broader than normal, that some of those broad waves really consisted of two component waves, the first a small wave, the second a larger one, and that the interval between the latter and the succeeding carotid wave was exactly equal to the *a*—*c* interval in other parts of the tracing. He, therefore, concludes that the second and larger elevation of the compound wave represented the true auricular wave, and that the smaller elevation, which preceded it, could only be ascribed to the contraction of the muscular fibres of the superior vena cava.

In Figs. 1, 3, and 5, as well as in the diagrammatic schemes constructed therefrom (Figs. 2, 4, and 6), the rhythmical occurrence of the wave *s* is readily observed. A point, to which we would particularly draw attention, as it is one which has not been hitherto demonstrated in the human heart, is the excessive length of the interval between the contraction of the fibres of the superior vena cava and the succeeding auricular contraction. This interval, the *s—a* interval, is of almost constantly uniform duration throughout the tracings, being equal to 0·6 of a second, though sometimes slightly shorter or longer. The excessive length of the *s—a* interval can only be explained by a depression of conductivity of the fibres passing from the superior vena cava to the right auricle. We are confirmed in this belief by our analysis of tracings taken both before and after the administration of $\frac{1}{50}$ gr. of atropine. Whereas the *s—a* interval was equal to 0·6 of a second before that drug was given, 11 minutes after its administration the interval was only 0·4 of a second, half-an-hour after the atropine, the *s—a* interval was 0·52 of a second, and, after one hour, that interval was again equal to 0·6 of a second. Comparing these results with those of the conductivity of the auriculo-ventricular bundle of the same case, we find that the improvement in the conductivity of the fibres between the vena cava and the auricle on the one hand, and of those of the auriculo-ventricular bundle on the other, was induced by atropine simultaneously, and that as the transient benefit, occasioned by that drug, passed off, the conductivity of both sets of fibres fell gradually and simultaneously to the former level.

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[1] "*Elementa Physiologiae Corporis humani*," 1757, tomus 1, pp. 399 and 410.

[2] "*Elements of Physiology*," trans. by W. Baly, 1838, Vol. I., p. 172.

[3] *Proc. Roy. Soc.*, London, June, 1876, Vol. 25, p. 172.

[4] *Jour. of Exper. Med.*, Vol. VII., 1905, pp. 690, 691.

[5] *Archiv f. Anat. u. Phys.*, Phys. Abth., 1906, S. 331.

The condition of the blood-pressure in cases of heart-block has not as yet received much attention. About two years ago, certain facts bearing upon this aspect of the condition were mentioned by one of us,[1] and the statement was amplified last year.[2] During the careful observations which were made on the patient, whose case is fully detailed in the present paper, the blood-pressure was repeatedly observed. The highest systolic pressure recorded at any time was 190 mm. Hg., and, with this maximum systolic pressure, the mean diastolic pressure was 80 mm. Hg. At other times, with a systolic pressure of 175, the diastolic pressure was 75 mm. Hg. Here, therefore, as was stated in the contributions already

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[1] *Edin. Med. Journ.*, 1905, Vol. XVIII., New Series, p. 9.

[2] *Brit. Med. Journ.*, 1906, Vol. II., p. 1113.

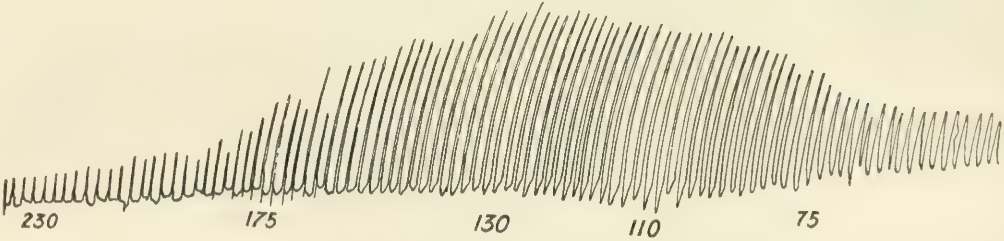

Fig. 7.—*Tracing taken from the patient with Erlanger's instrument, and slow release of air.*

referred to, the important point lies in the enormous variation between systolic and diastolic pressure. In one patient, who has been long under the observation of one of us, the systolic pressure is sometimes as high as 270, and the diastolic pressure only 80 mm. Hg. It is obvious that the long pauses, occurring between the contractions of the ventricles, must allow a great fall of pressure to take place in the intervals between the contractions.

As a rule, the estimations of the blood-pressure were made by means of the Riva Rocci, or the Erlanger sphygmomanometer. For ordinary daily observations, the former instrument is amply sufficient, as it is at once easy of application and accurate in results ; but when a permanent record is required, the instrument devised by Erlanger is essential. A description of the instrument has been published by Erlanger,[1] and a capital account, both of the instrument and of its method of employment, is to be found in the work of Janeway.[2] The mechanism resembles that of most of the mercurial sphygmomanometers with circular compression of the arm. The air contained in the pad and the tube leading to the column of mercury is, by a most ingenious valve, brought into communication with a tambour, whose lever inscribes its movements upon a revolving cylinder. The instrument is employed, in the same manner as the Riva Rocci and other syhygmomanometers, by increasing the pressure in the circular pad until the radial pulse is entirely obliterated, and then allowing the air to escape. It is found that, just at the instant of time when the radial pulse reappears at the wrist, the lever begins to manifest its largest excursions. The maximum systolic pressure is, therefore, easily determined. The diastolic pressure is not so definite, but it is believed that, just at the moment when the largest amplitude of the excursions begins to diminish, the diastolic pressure may be considered as present. The air may be allowed to escape slowly or quickly, the former giving the more accurate results. Four tracings, obtained from the patient whose case has been described, with the slow release of the air, are represented in Figs. 7, 8, 9, and 10, and another

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[1] *The Johns Hopkins Hospital Reports*, 1904, Vol. XII., p. 53.

[2] *The Clinical Study of Blood-pressure : a Guide to the Use of the Sphygmomanometer*, 1904, p. 93.

Fig 8.

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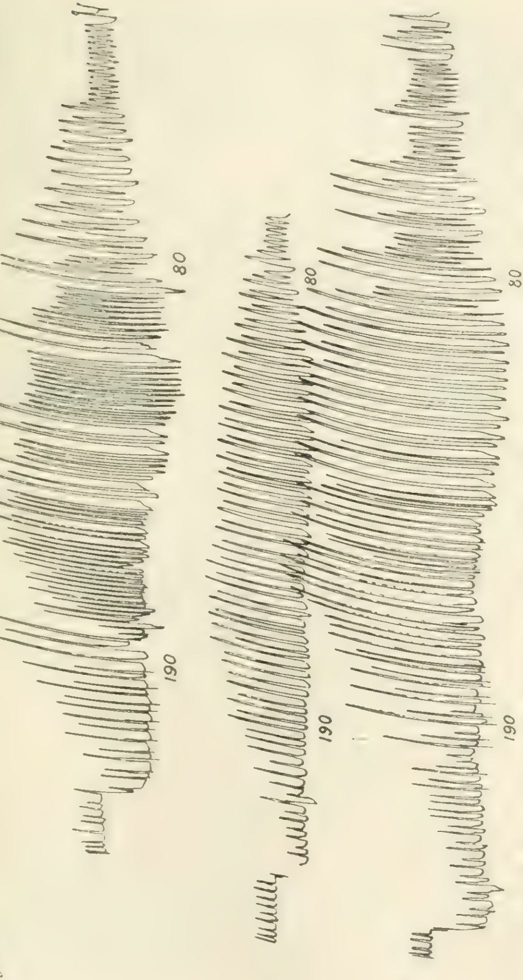

Figs. 8, 9, 10.—*Tracings taken from the patient with Erlanger's instrument, and slow release of air.*

tracing, taken with the quick release of the air, is shown in Fig. 11. In Fig. 7, the readings of the column of mercury are given at five different spots, the systolic pressure being 175, and the diastolic pressure 75 mm. Hg. In Figs. 8, 9, and 10, only the systolic and diastolic pressures are noted. In Fig. 11, the systolic pressure is 170, and the diastolic pressure 70 mm. Hg.

Sphygmographic tracings of the pulse are shown in Figs. 12 and 13. Fig. 12 gives the ordinary pulse manifested by the patient with a pulse rate of 28, which shows a type closely resembling the pulsus bisferiens, as it is often termed. Fig. 13 is extremely interesting, as it shows the character of the radial pulse after the patient had been treated with atropine applied locally to one of his eyes for severe iritis. The pulse rate, at this time, rose to 74 for a few days. Unfortunately this state of matters occurred during a holiday season, when neither of us was present, and no multiple tracings were obtained of the venous, arterial, and cardiac movements.

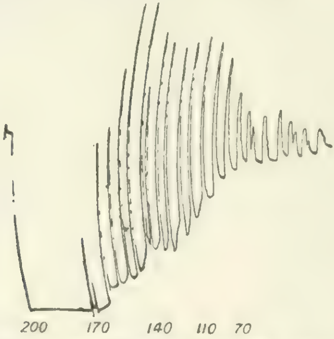

Fig. 11.—*Tracing with Erlanger's instrument and rapid release.*

It may be of interest to refer very briefly to the facts of the most recent instance of heart-block which has been under our observation. A mason, aged 53, was sent into Ward 29 of the Royal Infirmary, suffering from shortness of breath and general weakness. But little could be ascertained as to his hereditary antecedents. As a younger man, he served in the Army, and he used to be much addicted to alcohol, but for some years had been a temperate man. He had been exposed to climatic vicissitudes of the most pronounced degree, but he had never really, to his knowledge, suffered from any serious illness. The present condition of matters began about five years ago, and, with slight variations, had become progressively worse. On admission, he showed marked orthopnœa and considerable cyanosis. The rate of the pulse was from 80 to 90. It was perfectly regular, and almost equal. The apex beat was diffuse, and the heart somewhat enlarged, the right border of the heart being 2 inches, and the left border 5 inches, from midsternum. On auscultation, a galloping rhythm of the sounds was constantly present in the mitral area as well as in the tricuspid area. The first sound was followed by systolic murmurs; the second sound was almost invariably doubled, and the aortic element was greatly accentuated. A rough systolic murmur, obviously of aortic origin, could be heard over the upper portion of the manubrium. The arterial walls were markedly degenerated, distinct calcareous rings being distinctly felt. The systolic pressure was 195, and the diastolic pressure 125 mm. Hg.

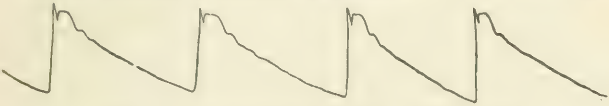

Fig. 12.—*Sphygmographic tracing. Ordinary rate.*

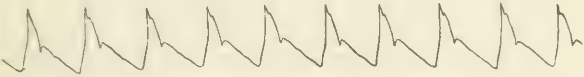

Fig. 13.—*Sphygmographic tracing after atropine.*

There were some evidences of hyperæmia at the bases of the lungs; the liver was somewhat enlarged, but there was no ascites; albuminuria was constantly present. The erythrocytes reached 5,100,000, the leucocytes 11,700, and the hæmoglobin was 80 per cent. The patient was under observation for four months, and, in spite of every method of treatment which could be thought of, he became progressively worse. During the last three weeks of his life, the rate of the pulse, which had almost invariably been above the normal, showed some tendencies to become subnormal, and, during the last week, it fell to 36 per minute. It remained for two days at, or about, this rate, and then oscillated between 44 and 68, which was its rate almost up to the time of his death. During the period of the great infrequency of the pulse, the patient was so seriously ill that it was impossible to think of troubling him with any attempt to obtain tracings, and we are therefore unable to give any graphic records of his condition. The patient frequently had attacks of Cheyne-Stokes breathing, most of which were characterised by periods of respiration of 30 seconds or thereabouts, while the intervening periods of repose were of almost equal duration. It is of interest to notice that, during the period of respiration, the maximum systolic pressure was usually about 200 mm. Hg., while, during the period of repose, it was usually about 195 mm. Hg. The diastolic pressure always seemed to be 125 in both phases, but it must be remembered that the criterion for this is not so definite as in the case of the systolic pressure.

After death, it was found that there were no lesions of the cusps, but simple dilatation of the mitral and tricuspid orifices with dilatation also of the aorta. The most interesting point lies in the fact that, on careful dissection of the auriculo-ventricular band, it was found to pass into a mass of fibrous tissue. A full microscopic investigation of the condition will be undertaken in the future, and we need only mention, at present, that this case adds another fact to the rapidly accumulating body of evidence in favour of disturbance of cardiac rhythm by means of a lesion in this region.

For much help in the clinical investigation of these two cases, we desire to thank Dr. J. C. D. Allan, who was untiring in his observations, and we also wish to express our obligations to Dr. E. B. Jamieson for his kindness in making a beautiful dissection of the heart in the second case.

SOME FRACTURES ABOUT THE ELBOW JOINT.

By ALBERT CARLESS, M.S.Lond., F.R.C.S.,
Professor of Surgery in King's College, and Surgeon to King's College Hospital.

[With Plates XII.—XVII.]

No one can have had much experience in surgery without having had brought to his notice a considerable number of cases in which serious limitation in the movements of the elbow joint has resulted from an injury, and he must have been impressed with the great discomfort, and often the real loss of utility and power, which follow such a lesion. Fixation of the knee or hip joints involves a certain degree of difficulty in progression and of discomfort in sitting ; with the shoulder and wrist joints there is always possible a considerable development of compensatory movement in adjacent articulations, but with the elbow there is no possibility of compensation, and fixation here means much disability and loss of usefulness. On investigation, it will be frequently found that the trouble dates back to an injury in the neighbourhood of the joint in childhood, and it is with the idea of emphasising the value of operation in the treatment of some of these cases, that this article is written.

The elbow joint is not altogether a simple piece of mechanism of a hinge type ; for, whilst this is the primary movement, other possibilities have been developed. In the first place, the lower articular surface of the humerus is not directed exactly at right angles to the axis of the bone, but the inner condyle and trochlear surface are somewhat elongated, so that the axis of the ulna is thrown slightly outwards to a varying degree. This carrying angle, as it is termed, between the axes of the humerus and ulna, is generally about 10° or 15°, and is obviously existent for two reasons : first, to enable the arm, when hanging, to clear the projection of the iliac crest, and, secondly, to carry the hand to the mouth when the elbow is flexed, without other rotary movements of the shoulder. Then, too, it must be remembered that the superior radio-ulnar joint communicates with the elbow, the radius which carries the hand working on the ulna as a pivot

The treatment of any injury about the elbow should be directed towards the perfect restoration of both these conditions, and although it is not infrequently impossible to attain to such a desired end, yet no treatment is really satisfactory which fails to attempt it. Modern surgical progress has not left the question of fractures and their treatment in the place as it was thirty or more years ago. Diagnosis has been improved by the introduction of radiography, whilst treatment has been placed on a totally different footing by a recognition that prolonged immobilisation and rest in plaster of Paris is not the do-all and end-all. The value of massage has been demonstrated, and the fact that early mobilisation is not always undesirable has been accepted ; moreover, the safety of modern aseptic operations has removed our fears of transforming a simple into a compound lesion.

Whilst rejoicing in the assistance that has been given to questions of diagnosis by the the introduction of radiography, one cannot but fear that, at times, too much reliance is placed on it, and that the art of diagnosis by digital examination is being lost. In quite a large majority of cases, some shrewd idea as to the nature of the lesion ought to be possible, even if the swelling of the arm has attained to such dimensions as to render a complete diagnosis impracticable ; then radiography correctly comes to our assistance to determine the exact extent of the injury. Two or three points in reference to radiography must not, however, be forgotten if grave errors are to be avoided. In the first place, one must never depend on a skiagraph taken only in one direction : the most deceptive ideas may, in this way, be conceived as to the nature of the injury. This is particularly true in regard to fractures of the shafts of long bones. Then, every such picture is necessarily an exaggeration, the degree of which varies with the square of the distance between the bone and the plate ; it is quite possible that a displacement, which looks alarming in a skiagraphic picture, is in reality not of much importance. Moreover, much depends upon the position from which the picture is taken : an apt illustration of the errors, which may creep in from non-recognition of such a point, is afforded by Fig. 1. A small fragment nearer the lamp may cast a shadow, which will completely overlap that given by a larger fragment nearer the plate, and a totally wrong impression as to the character of the lesion may result, unless it is corrected by pictures taken in a different direction, or by the application of stereoscopy. Finally, one must not forget that cartilage throws no shadow, and therefore lesions to the epiphyses, before ossific centres have developed, are unrecognisable by skiagraphy. In examining an arm, with a view to diagnosis, the most important points to be noticed are :—(*a*) The relative position of the bony points to each other, and the distances of the inner and outer condyles from the olecranon ; (*b*) the respective lengths of the arm and forearm, as measured from the condyles to the deltoid tubercle on the back of the acromion process above, and to the styloid processes of the radius of ulna below ; (*c*) the existence or not of crepitus, its character, and location ; and (*d*) the mobility or not of portions of the bones. In interpreting skiagraphs in children, the arrangement and date of appearance of the ossific centres in the lower epiphysis of the humerus must be kept in mind. That for the capitellum, or radial head, appears first at the age of 3 years, whilst the centre for the trochlear surface does not develop until the 8th year ; these two coalesce at about the age of 12 or 13 years, and join the shaft at about 17. The ossific centre for the internal epicondyle manifests itself at about the age of 5, and remains distinct after the main epiphysis has united with the shaft, viz., until the 18th or 19th year. The external epicondyle is small and of but little significance ; it appears as a distinct centre at about the 13th year, and joins the shaft about 4 years later. The olecranon is chiefly developed from the shaft, and there is but a small ossific centre at its upper end, which appears at about the age of 10, and unites with the shaft at about the 15th year. The head of the radius consists of cartilage until the 6th year, when a thin ossific plate appears in it ; the main portion of the head is, however, developed from the shaft, and the thin plate-like ossific centre unites with it at about the age of 16 or 17.

The account of some of the fractures in this region, which follows, does not claim to be exhaustive, but merely deals with some which have come under the recent notice of the writer, and are suggestive of the value which may be attached to operation in their treatment. Transverse fractures of the lower end of the humerus are not uncommon lesions at all ages, but their character varies somewhat with the age. Three chief forms may be described :—

1. *Transverse Supracondyloid Fracture* may occur in children or adults as the result of falls on the elbow, or, indirectly from falls on the outstretched hand, whilst the elbow is usually in the flexed position. The displacement of the lower fragment is either forwards or backwards, generally the latter, and the character of the lesion, its diagnosis from backward dislocation of the elbow, and its treatment, are generally recognised. Only two points need notice in this connection :—(1) that the line of the lesion is well above the joint, and, therefore, passive and active movements are not necessary at as early a stage as if the joint were involved; and (*2*) that the internal angular splint is a very inefficient contrivance for treatment. The common form with displacement backwards is generally replaced with ease by bending the arm, and making traction from the hand, whilst the olecranon is pushed downwards and forwards by the thumbs. A posterior gutter splint of poroplastic or plaster of Paris is then moulded to the limb, and a short anterior splint may be conjoined with it. Operation is seldom required in uncomplicated cases.

This form of fracture is sometimes associated with a vertical cleft, or split, which runs down into the joint in the shape of a T or Y. The fragments, by this means, are separated one from the other, but, if the periosteal envelope is not torn, displacement does not necessarily ensue. If, however, the periosteum is badly lacerated, they may be displaced inward and outward, and some broadening of the elbow will result. A case of this sort should always be submitted to operation in order to fix the fragments one to the other, even if it is unnecessary to secure the united fragments to the shaft. It is probable that the introduction of a screw transversely through the fragments will suffice to fix them satisfactorily, and then possibly flexion to a little more than a right angle, and the use of antero-posterior splints will suffice to bring the case to a satisfactory conclusion. In other cases, the comminution of the lower end of the humerus may be more extensive, and, indeed, it may be crushed into small fragments. If such a condition is left, it is quite possible for union to occur; but it will be associated almost certainly with the development of much callus, which will obliterate the olecranon and coronoid fossæ, besides being accompanied by intra-articular adhesions of such a character as to interfere permanently with the movements of the joint. It is probable that, in such cases, sub-periosteal resection of the lower end of the humerus will be the best treatment that can be adopted, if it is found, on exploration, that the fragments cannot be fixed to one another satisfactorily by screw, nails, or pegs. The recorded results of such operations have been encouraging.

2. In infants and young children, a *complete separation of the lower epiphysis* is occasionally seen. Before the age of three years, this fragment is purely cartilaginous, and will include both condyles and epicondyles. The line of cleavage is well above the joint, so that the lesion is extra-articular. Radiography shows nothing except the backward displacement of the ulna and radius.

3. Much more common than this is the so-called *separation of the lower epiphysis in older children*, which is an accident frequently observed. In it the line of cleavage generally extends into the diaphysis on one side or the other, so that the lesion is partly a separated epiphysis, partly a fracture. The extent and direction of the portion of diaphysis detached are very variable. Sometimes the line of fracture runs cleanly and obliquely inwards or outwards above the epicondyle; but not rarely the fragment does not include the whole antero-posterior thickness of the shaft, the posterior aspect being encroached on more extensively than the anterior. The upper edge is sometimes very irregular and spiculated, and, in some cases, an angular wedge-shaped portion is torn out, leaving a triangular cavity behind; the exact replacement of such an irregularly shaped fragment is not easy even by operation. My experience quite accords with that of Cotton (*Annals of Surgery*, February, 1902), that the obliquity is almost always downwards and forwards; the opposite type of obliquity described by Kocher must certainly be extremely rare, if it occur at all.

In spite of the encroachment on the diaphysis, the general features of an epiphyseal separation are largely maintained. Thus the periosteum remains firmly attached to the epiphyseal cartilage, but is stripped off the diaphysis for a considerable distance upwards, and in the cavity thus formed, blood collects, constituting an important element in the swelling which almost always follows this injury. The displacement is generally backwards (Fig. 1), but occasionally forwards. In the former circumstances, the lower end of the diaphysis projects forwards, and constitutes a swelling just above the crease of the elbow, which sometimes threatens to perforate the skin, either on the inner or outer side, according to the degree of lateral displacement. Thus in Fig. 1 the skin on the outer side was nearly torn. In such cases, the detached periosteal envelope is punctured by the end of the diaphysis, and the projecting fragment of bone is grasped by the periosteal cuff, and reduction may be thereby hindered. The elbow joint itself is rarely involved in this lesion, although in older children, it is rather more liable to injury than in the younger, owing to the downgrowth of the diaphysis on the inner side, whereby the internal epicondyle is separated from the rest of the epiphysis.

Considerable difference of opinion has arisen as to the effective treatment of these cases of epiphyseal separation.

In the first place, there has been not a little discussion as to whether they should be treated in the flexed, or extended position of the arm. Not a few surgeons of note have recommended and practised the latter, including Liston, Bardenheuer, and König; but the general trend of opinion is in the opposite direction. It is difficult to see how the backward tendency of the lower fragment is to be satisfactorily corrected, when the forearm is fully extended, and the experience, that one has gained by operating on these cases, certainly goes to prove that flexion is the correct position to adopt.

This being granted, two questions arise: What angle should be adopted, and how should the flexion be maintained? Personally, I have not the slightest doubt that the best position is that of acute flexion, knowing that it is subsequently more important to gain complete flexion than complete extension; slight limitation in the latter movement is less serious a sequela than impairment in the power of flexion. Experience proves that there are certain cases, which can be readily reduced into position, with or without an anæsthetic, and in which full flexion can be easily secured. Thus

PLATE XII.

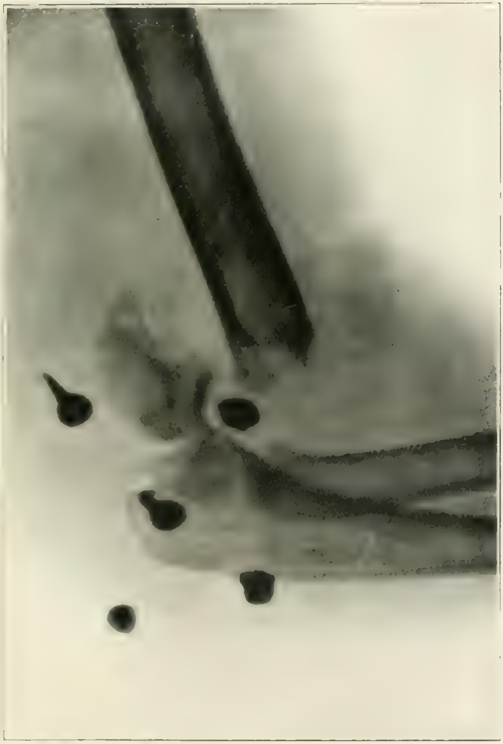

Fig. 1.—*Fracture of lower end of humerus in a child of seven years with displacement of the fragment backwards. The ossific centre for the capitellum can be seen overlapping the shadow cast by the upper end of the ulna, and separated by an interval from the displaced portion of the diaphysis. The five dark spots are tin tacks in the internal angular splint which was upon the arm when sent up to hospital.*

PLATE XIII.

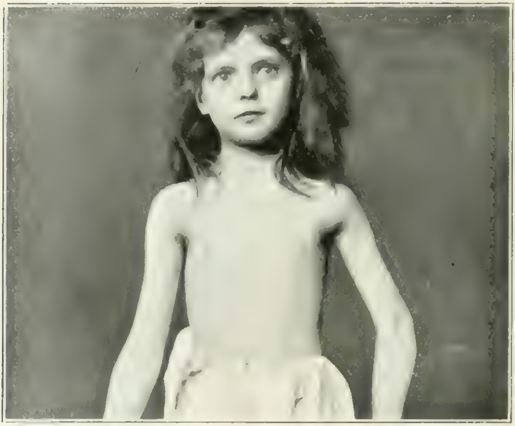

Fig. 2.—*Old injury to left elbow. The condition of Cubitus varus can be contrasted with the normal carrying-angle of the right arm.*

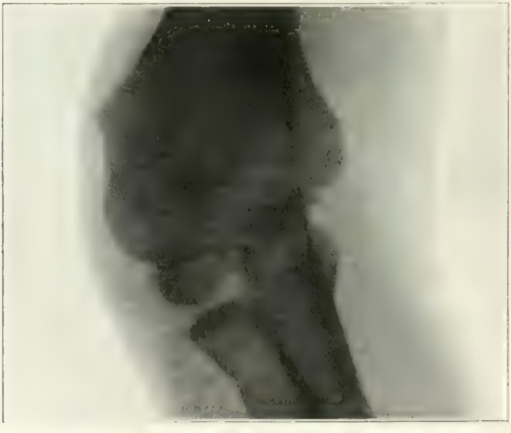

Fig. 3.—*Skiagraph of left elbow of child with Cubitus varus, seen in* Fig. 2. *The internal epicondyloid thickness is distinct in the original, and the epiphyseal line at the lower end of the shaft of the humerus is set obliquely to the axis of the bone.*

when the patient is seen early, and before much swelling has occurred, it is often a matter of the greatest ease to reduce the fragment of bone. Traction is made upon the forearm which is gently, but forcibly bent, whilst pressure upon the olecranon forwards and downwards carries the fragment into place. Then arises another question. Should the forearm be kept prone, or supine, or half-way between? The last is the more comfortable position, but any degree of pronation is likely to be associated with some tendency to cubitus varus, and the restoration of the carrying angle is more likely to be secured by keeping the forearm supinated. Some authorities, *e.g.*, Scudder, recommend that the arm should be bound to the side in a position of full flexion; but the advisability of such a practice is doubtful, as it is likely to draw the forearm inwards. It is wiser to flex the forearm acutely with the hand supinated, and bandage it to the arm so that the hand is approximated towards the shoulder. After its fixation in this position, rotation inwards from the shoulder may be allowed, and the arm then bound to the side. The adoption of such a procedure as this would prevent the development so frequently of the ugly gun-stock deformity. The arm should be kept in this position for 12 or 14 days, and then placed in a hollow poroplastic gutter splint, hinged at the elbow in such a way as to reproduce the carrying angle. The amount of movement is graduated by tapes passed through holes on either side of the upper and lower segments; the range of movement is gradually increased, and, in this way, it is possible to gain excellent functional results.

When, however, the case is not seen until considerable swelling has taken place, it is often impossible to effect reduction, and the adoption of the acutely flexed position is impracticable. It is in these cases that operative treatment is desirable. During the last few months, I have seen a number of such cases, and have been much impressed by the facility with which reduction could be obtained by a very simple operative procedure, and I have no hesitation in recommending this in suitable cases. The operation, in the majority of instances, consists merely in cutting down to the site of the fracture on either side, dividing the periosteum, so as to allow the blood collected beneath it to escape, and then manipulating the fragment into position, and putting up the limb in acute flexion. Care must of course be taken to avoid vessels and nerves, and this can often be ensured by limiting the use of the knife to the division of the skin and subcutaneous structures over the inner and outer condyloid ridges, a pair of sinus foreceps being then introduced through the deeper structures, including the periosteum; separation of the blades of the forceps will generally suffice to make an opening large enough to introduce the finger. The blood escapes, and bilateral digital examination will permit the condition of affairs to be recognised, and a little traction and manipulation will ensure replacement. Difficulty sometimes arises owing to the irregular edges of the fragment, and, in one or two cases, I have had to clip off projecting spicules of bone; this is undesirable, if possible, as the irregularities help to maintain the fragment *in situ*, when once the displacement has been overcome. The adoption of the acutely flexed position usually suffices to maintain apposition, and the introduction of retentive appliances, such as wire, or screws, or nails, is generally superfluous. The lateral incisions are closed by a stitch or two, and the limb is put up in accordance with the rules suggested above. The after-treatment is exactly the same.

Of course the risk of sepsis has to be considered, but with careful precautions this is now a rare occurrence. In only one of my cases have we had any trouble from this cause, and in it there was much swelling before the operation, the skin over the ante-cubital fossa was bruised, swollen, and blistered, owing to the pressure of the bone and effusion, and it is quite an open question whether suppuration would not have occurred apart from operation. In the other cases, one has not been always able to obtain a complete restoration of the carrying angle, nor even always of the antero-posterior movements: but the results have been so gratifying that one has no hesitation in recommending this procedure, and in practising it whenever a suitable opportunity has offered.

Of course one is fully cognisant of the fact that very excellent results have been obtained in favourable cases as the outcome of extreme care and persistent massage and movement, and this even in cases when operation has been recommended to, but declined by the patient; but the time and trouble that have been necessary to gain such an end, and the amount of pain and discomfort that have been experienced are out of all proportion to the slight discomfort and risk associated with operation. In cases, however, in in which such minute attention to detail is not possible, as in many out-patient casualties, there can be no question as to the value of treatment by operation.

The bad results of imperfect treatment are many and various.

1. Limitation of movement may involve either the hinge action of the elbow, or the rotation of the superior radio-ulnar articulation. It may arise from the presence of intra-articular adhesions, fibrous or osseous: from the filling up of the olecranon or coronoid fossæ by callus, or by the actual displacement of bony fragments. Thus, in Fig. 1, it is obvious that unless the lower fragment of the humerus is restored to place, the olecranon will necessarily hitch against the lower end of the humerus, whilst the ulna must come in contact with the callus, formed about the lower end of the shaft, and thereby flexion be limited.

2. Non-restoration of the carrying-angle is a result which is often seen and cannot always be prevented. The result may be that the forearm and arm are quite straight in the same axis, the so-called "gun-stock deformity": or there may be a definite angular deformity, known as cubitus varus, or valgus, as the case may be. Fig. 2 represents a girl of ten years with a well-marked *cubitus varus*, the result of an epiphyseal lesion some years previously. The skiagraph (Fig. 3), quite plainly shows the epiphyses for the capitellum and trochlear surface, still distinct from each other, whilst the internal epicondyloid centre is separated from the shaft by an oblique line of cartilage, which is placed in its normal direction. The line joining the two condyles is quite obviously at right angles to the axis of the shaft, but, interposed between the inter-condyloid line and the epiphyseal line is an angular mass of newly-formed bone, which deflects the axis of the forearm inwards. The discomfort of such a condition is quite evident when one looks at the photograph of the child. In this case, an angular wedge of bone had to be removed in order to restore the arm to its normal direction.

Cubitus valgus is the opposite deformity, and is to be looked on as an exaggeration of the normal carrying angle. It also usually results from imperfect treatment of an epiphyseal separation involving the lower end of the bone. The dependence of these two deformities upon such transverse lesions is emphasised by Cotton (*op. cit.*), who investigated the origin of a considerable number of them. In the valgoid condition, the chief trouble often arises from the fact that the inner condyle is unduly large and prominent, and carries with it the ulnar nerve which is thereby exposed to injury. A case of this sort came under my notice not long ago for this traumatic neuritis, which was recurring constantly as the result of slight injuries.

3. Imperfect development of the bones may result from these epiphyseal lesions if they are not correctly restored to position.

4. Paralytic conditions, resulting from nerve lesions, are also not unknown. The ulnar nerve is exposed to injury in any fracture, which involves the inner condyle, whilst the displacement backwards of the lower fragment causes undue pressure anteriorly by means of the lower end of the shaft, and the median nerve may be thereby involved. Mühsam has laid stress on these lesions in a recent communication (*Zentralb. für Chirugie*, 1906, No. 32, p. 877).

The so-called *Ischæmic contracture* is also occasionally seen to follow these lesions, but it must be clearly understood that it is not due to nerve pressure or to splint pressure, but, as originally described by Littlewood, of Leeds, is due to an intrinsic shortening of the muscles of the forearm resulting form a fibrosing type of myositis. A case of this type was seen by me a little time since, in which no splints had been used which could exercise pressure upon the forearm.

Fracture of the External Condyle, or, as it is sometimes termed, Epicondylo-capitellar fracture, is another lesion about the elbow, which requires early operation in order to gain good results. The outer is more frequently broken off than the inner condyle, probably owing to the fact that it is more exposed to direct injury. The old idea that it was broken, owing to the radius being separated by some little distance from it, and, therefore, a fall on the hand might drive the

PLATE XIV.

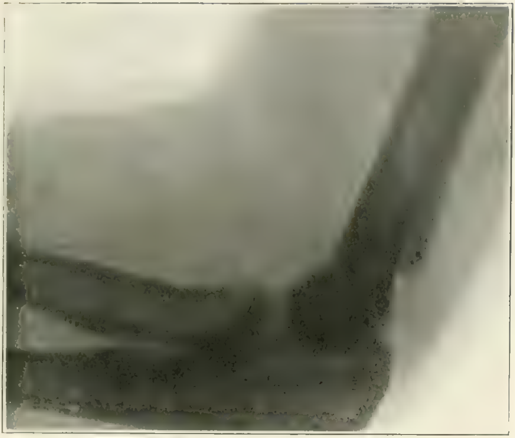

Fig. 4.—*Fracture of external condyle of humerus (side view).*

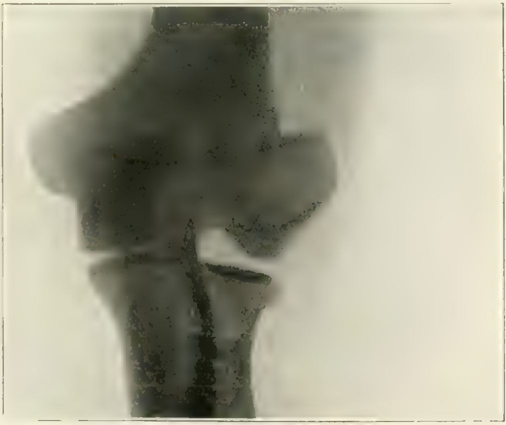

Fig. 5.—*Fracture of external condyle (antero-posterior view).*

PLATE XV.

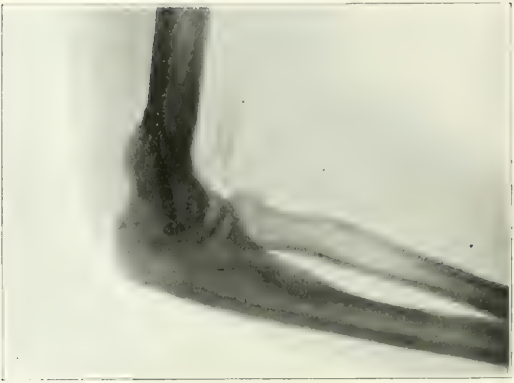

Fig. 6.

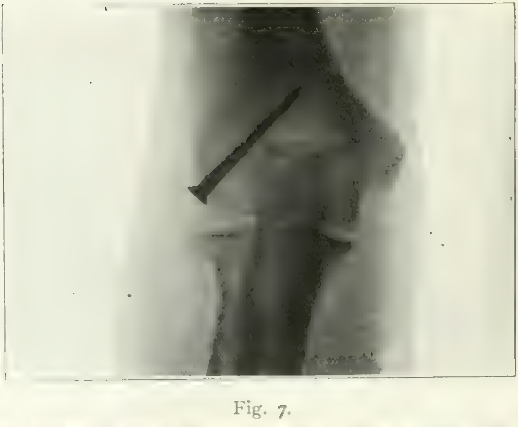

Fig. 7.

Figs. 6 and 7.—*Fracture of external condyle after operation. The screw whereby the fragment was fixed can be seen in situ from the side and front respectively.*

radial head up against it with such force as to break it off, is scarcely worthy of credence; as a matter of fact, the injury is usually the result of direct violence. The line of fracture runs into the elbow joint, and usually reaches the joint between the trochlear surface and the capitellum; the fragment thus detached is drawn slightly downwards and rotated forwards by the muscles attached thereto. Crepitus can generally be obtained without much difficulty, but the arm becomes rapidly swollen, so that reposition, without an operation, is almost impossible. Acute flexion might enable the fragment to be put into place, but it would always be a matter of the greatest uncertainty as to whether it would remain there. Imperfect union of this lesion might lead to modification of the carrying angle, but chiefly to imperfect flexion of the joint, owing to the anterior projection of the fragment. There can be no question as to the desirability of fixing the fragment by operation. The following case well illustrates this method of treatment:—

A. C., a school girl, aged 15 years, seen with Dr. George Thomson, of Ealing, was riding to school on her bicycle two days before I saw her, when her machine skidded, and she fell heavily on the right elbow. Severe pain was complained of, and the elbow soon became much swollen. She was examined under an anæsthetic; the olecranon was intact; there was no transverse fracture of the humerus, but distinct crepitus was obtained over the external condyle. Fracture of this process was diagnosed, and the arm was put up in a position of acute flexion.

Two days later, a radiographic examination was made, and the two radiographs here reproduced (Figs. 4 and 5) were obtained. In Fig. 4, taken from the side, there is but little indication of the fracture, except that there is an abrupt and irregular termination to the outline of the outer condyle (the apparent posterior border of the bone in the skiagraph); but the detached fragment cannot be seen, owing to the overlapping of the shadow cast by the rest of the bone. The anterio-posterior view (Fig. 5), however, gives a very different aspect of affairs, and yet the skiagraph is a most deceptive one, and might easily lead to an entirely wrong conception of the displacement. It appears as if the outer condyle and capitellum had been broken off and displaced bodily outwards; but no projection on the outer side was to be felt, and indeed no such external displacement was present. Had this been the case, a gap in the continuity of the articular line of the humerus would have been visible, but this is absent. The actual state of affairs was that the upper end of the fragment projected forwards in a plane anterior to that of the shaft, and the skiagraph being taken with the lamp slightly to the inner side of the axis of the limb, the shadow of the fragment overlapped and projected beyond that of the underlying shaft. This is a useful illustration of one of the difficulties which surround the interpretation of skiagraphs by those who are not constantly engaged in dealing with them.

As the patient was desirous of becoming an expert violinist, it was essential that the movements of the arm should be restored perfectly, and also that it should be free from deformity; operation, was, therefore, proposed and accepted. A curved incision was made over the condyle, and the bony fragment was found to be loose, except for the muscular attachments below. Some little difficulty was experienced in fixing it *in situ* so as to allow the bone to be drilled; finally, it was secured in perfect apposition by means of a screw, as seen in Figs. 6 and 7. Considerable care had to be exercised in introducing the drill, so as to carry it upwards in the substance of the bone throughout its course; the humeral shaft is here thin antero-posteriorly, and a very slight error might have taken the screw into, or through, the olecranon fossa, and thereby interfered with full extension of the elbow. The wound was subsequently closed by buried sutures in the soft tissues, and an intradermic (Halsted) suture of silkworm gut in the skin. It healed perfectly, and the scar is now (six months later) but little evident. Passive movements were commenced after a fortnight, and complete restoration of the function of the joint in every particular has been obtained.

Fracture of the Internal Condyle and of the Trochlear Surface are much less common than the injury just alluded to; the displacement varies, but is generally one in which the fragment travels backwards and upwards, and perhaps a little

PLATE XVI.

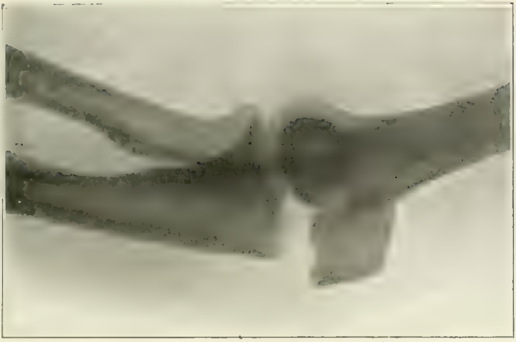

Fig. 8.—*Fracture of olecranon before operation.*

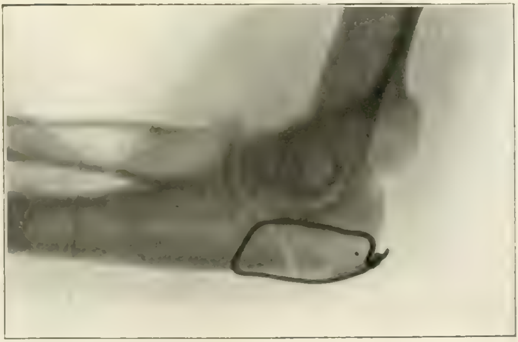

Fig. 9.—*Fracture of olecranon after operation. The interval can still be seen, and is occupied by soft bone.*

PLATE XVII.

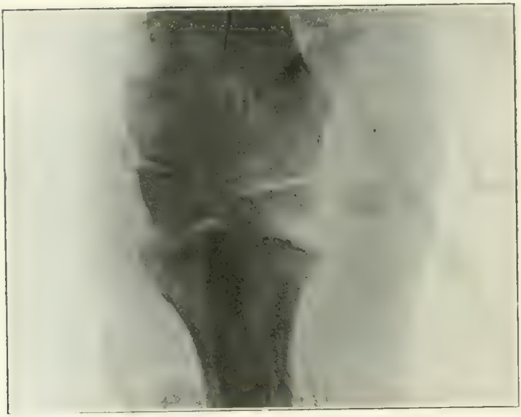

Fig. 10. *Separation of internal epicondyle with displacement downwards and inwards.*

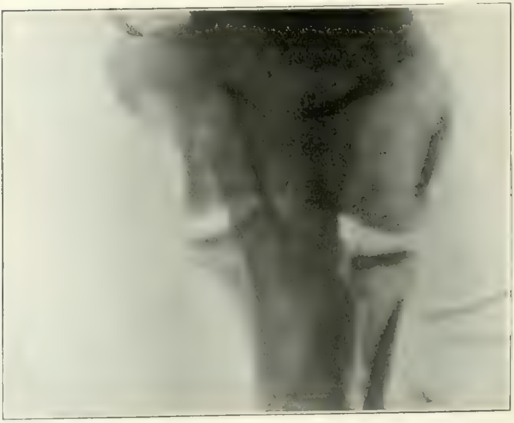

Fig. 11. *Longitudinal fracture through head and neck of radius.*

inwards (Poland). Seeing that this fragment carries the ulna in articulation with it, angular deformity of the forearm is only too likely to follow, and hence operative treatment, in order to secure complete apposition, would be most necessary. Unfortunately, no specimen of this injury has come to my notice.

Separation of the Internal Epicondyle is by no means a rare accident. The ossific centre, as already mentioned, appears about the fifth year, and forms part of the lower epiphysis of the humerus; but, after a few years, it becomes separated from the latter by the downgrowth of the shaft, so that, for the last five or six years in which it is seen, the epicondyle is distinct, and its base of attachment is set nearly at right angles to the articular surface. Direct injury is usually responsible for its separation from the shaft, and the most common displacement is downwards, owing to the drag of the muscles arising from it (Fig. 10). The rule usually given hitherto as to the treatment of this injury is to attempt replacement, by means of strapping with the arm flexed to a right angle, or even to excise the fragment, if reposition fails. Non-union is not necessarily associated with much disability, but the development of callus, or of fibrous scar tissue, may lead to involvement of the ulnar nerve, and consequent discomfort. It is quite clear that the correct treatment is by operation. A small incision will enable the fragment to be apposed to the shaft; and then it is easily fixed by the introduction either of a nail or of a screw. Cotton (*Annals of Surgery*, March, 1902) points out that there is usually but little interference with mobility in these cases, except possibly some slight loss of extension, but that there is probably some local deformity, though without any deviation in the axis of the forearm.

Fracture of the Olecranon.—It seems scarcely necessary at the present day to emphasise the fact that there is only one efficient method of dealing with fractures of the olecranon, when the periosteal, tendinous, and fascial tissues are involved to such an extent as to allow of complete separation of the fragment, and that consists in operation. The fracture is usually transverse, and situated near the base of the process; should there be no separation, immobilisation for a week or two to allow the parts to settle down, and then massage and passive movements to commence with, and subsequently active movements will suffice to obtain an excellent result. No splints are required, but the arm should be kept in a sling for a while.

When, however, there is distinct separation of the fragment, treatment by operation is most desirable. Apart from this, it is possible to gain satisfactory fibrous union, but the treatment is tedious and painful, and one cannot always ensure firm union. In some cases the fragment is drawn up, and becomes adherent to the shaft of the humerus, and a false joint is developed between the fragment and shaft of the ulna. Fixation of the fragment by operation (as, for example, in the case represented in the skiagraphs, Figs. 8 and 9) gives a certain and rapid restoration of function. It is perhaps best to employ silver wire for the purpose, and it is introduced so as to emerge on the fractured surface just short of the cartilage. Sufficiently firm wire should be employed to stand the strain of movements before the bone has united. It is, in many cases, allowable to permit gentle movements at the end of eight days after the operation, and, in the majority of cases, a full range of movements is practised at the end of 14 days. I have operated at all ages for this lesion, from 14 years up to over 70, and certainly, in the last case, the old lady was able to extend and flex her arm fully at the end of 14 days, and that without any pain.

Fractures of the Head of the Radius also require operation in order to secure favourable results if there is any separation of the fragments. Mere fissures of the bone can usually be left, and are treated by massage and early mobilisation; but if the head is comminuted, or when it is separated either by a transverse, or by a vertical fissure (*e.g.*, Fig. 11), then the only chance of gaining a good result, without permanent impairment of rotation of the limb, is by operation. Sometimes it may be possible to fix the fragment into position by silver wire; but, in the majority of cases, excision of the head will be required. Even if the fragment has been wired, there is often a great deal of reaction and some development of callus, which will prevent movement, and require excision later on.

ON THE USE OF OPIUM IN ACUTE AND CHRONIC DISEASES.

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If I were asked to name the best *single* test of the judgment and clinical capacity of a medical practitioner, I should reply that, in my humble opinion, it would be his skill in the use of opium—to know when and how to give it, and when to withhold it.

When examining candidates for diplomas at the Conjoint Board, I own I often felt alarmed at the possible consequences that might follow, if all those hypodermic injections of morphia, which were prescribed on paper, were actually introduced into the connective tissue of real patients, and I have noticed, not without consternation, what a large place in the resources of some practitioners is occupied by the hypodermic syringe. I feel convinced that a veritable history of all the doings of that small but potent instrument would make very sensational reading!

The first thing, then, to be kept in view, with regard to opium, is the necessity of great caution in its use and dosage, and especially in the hypodermic use of morphia. An overdose given by the stomach can be vomited, or, in part, washed out, but, once introduced into the connective tissue under the skin, it can never be withdrawn.

It should be remembered that, when administered in this way, it enters the circulation very rapidly—this, indeed, is one of the advantages of the method, as it enables the practitioner to relieve suffering with great rapidity—but it has its drawbacks; one of these is the temptation it offers the patient to self-administration and the establishment of the morphia habit, another is the risk, with certain hyper-sensitive patients with *feeble hearts*, that morphia given hypodermically may cause *cardiac failure*. The feeble, degenerated cardiac muscle is readily paralysed by morphia, save in quite small doses, which should be guarded, and combined with a cardiac stimulant.

The great use of opium is to *relieve pain*, in whatever way produced. Nowadays, we rarely use opium as simply a hypnotic, but if insomnia is dependent on the existence of *pain*, then we may be compelled to give an opiate for its relief.

We can now have recourse to a great number of hypnotics, which were quite unknown a generation or two ago—*e.g.*, bromides, chloral, chloralamide, sulphonal, trional, tetronal, veronal, and many others.

Nor are we so dependent on opium for the relief of pain as we were formerly; we have now numerous analgesics, better suited than opium for the treatment of many painful affections. Salicine and the salicylates have, to a great extent, superseded opium for the relief of pains of rheumatic origin.

Antipyrin, phenacetin, pyramidon, and a host of related drugs, have, in many instances, taken the place of opium as a "*pain killer*." In this respect, the field of usefulness of opium has been, in modern times, greatly restricted. But some of these, synthetically produced analgesics, have proved to be very dangerous agents—far more so than opium, owing to their destructive or paralysing actions on the red-blood corpuscles. I have seen more than one fatal result follow their incautious use, and many more have been reported in the medical journals. These fatal accidents have generally been caused by an excessive dose, given before the toxic action of these new drugs had been fully investigated.

I would say, in this connection, that it is always a good plan to avoid being amongst the first to use a new drug, wait until its safe dosage has been accurately made out, and wait to see if any unsuspected by-effects of a toxic nature are to be feared.

Where opium stands unrivalled as a therapeutic agent is in the relief of *pain accompanied by spasm*; no known drug approaches it in efficiency for this purpose. And these are the most severe and terrible form of pains which we are called upon to treat.

Take, for instance, the various forms of *colic*—intestinal, biliary, and renal—attacks of spasmodic asthma—the pain of angina! What could adequately replace opium in the treatment of these affections? I have seen—and who has not?—the most severe paroxysm of asthma yield, in a few minutes, to the hypodermic injection of a quarter of a grain of morphine combined with a sixtieth of a grain of atropine. In some cases it is preferable to give morphine by the mouth, combined with some spirit of chloroform and aromatic spirit of ammonia. There is thus less risk of depressing the cardiac action in debilitated patients. With regard also to attacks of biliary colic, I prefer to give opium by the stomach, because I have found that it gives more complete relief when administered in this way. Indeed I have often had reason to think that the nearer to the seat of pain the opium is absorbed, the better and more lasting is the effect. Opium liniment rubbed into a painful joint, opium suppositories, a small opium enema in painful conditions of the rectum and bladder, friction of the abdomen with opium in lead colic, an opium lotion in painful conjunctivitis; these are a few instances of the local applications of opium usually attended with satisfactory results.

In giving opium by the stomach, it is by no means a matter of indifference what preparation of opium you use. It may often be observed that a preparation like Battley's solution will agree well with a patient who would be made sick and uncomfortable by a dose of laudanum. In the prescribing of opium, it is most important to bear in mind these differences in the action of different preparations and combinations. Some patients, who cannot take a dose of morphine by itself without being nauseated, will take it, combined with the aromatic spirit of ammonia, without any difficulty.

In treating the colicky attacks and the gastro-intestinal catarrh of children, you can rarely dispense with the use of small doses of opium. It is then best given *per rectum* mixed with a little thin starch or arrowroot, and I prefer to give it in the *solid* form—½ a grain to 2 grains or more of Dover's powder or the compound Kino powder, according to the age of the child. The opium in this form is absorbed slowly and there is no risk of rapid narcotism—as will sometimes occur when a fluid preparation is used—and it is necessary always to keep in view the extreme susceptibility to the action of opium of some young children.

An instance of the danger attending the incautious use of fluid opium I once saw in Hospital practice. A dram of tincture of opium was given *per rectum* in a case of typhoid fever, with diarrhœa and hæmorrhage, in a young adult; rapid narcotism set in, and a fatal result quickly followed. It is very important to exercise great caution in the use of opium in enteric fever, as the heart is often very weak, and yet opium is very frequently absolutely needed to keep the bowel at rest. The best and safest plan is to use solid opium as it exists in Dover's powder, which may be given in 5-grain doses (for adults) in small enemata (an ounce or two of thin arrowroot) which can be repeated, if well borne by the patient. It often acts extremely well, not only by checking excessive diarrhœa, but also by quieting the nervous system and promoting restful sleep.

The use of opium in acute peritonitis, and in acute cases of intestinal obstruction is a debatable subject. The teaching, once almost universal, that "opium should be your sheet-anchor" in such cases, is no longer in such favour as it was. Stated in that unqualified way, the teaching was, I think, unsound. It is certainly our duty, in such cases, to relieve severe pain, but the severity of the pain, having been subdued by the cautious and sparing use of opium, the occasion for its administration is at an end. It is decidedly bad practice to induce paralysis of the bowel, as was often done, by continued large doses of opium. Here, indeed, the local external application of opium in the form of a hot linseed poultice, freely sprinkled with laudanum, is free from the objection that might rightly be felt to giving opium internally. Opium and its preparations are often used for the relief of cough. But here again it must be handled with judgment and caution. As the object of cough, in many cases, is to get rid of excessive or tenacious secretion from the respiratory mucous membrane, which, if allowed to accumulate and block up the air-passages, would be fatal to the patient, we must beware how we attempt to arrest such a cough by giving opiates. To give a dose of opium in order to relieve the night cough in an aged person, with pulmonary emphysema and chronic bronchial catarrh, is to incur the risk of finding him suffocated by his own secretions in the morning. The same may be said with regard to the cough of a young child with broncho-pneumonia or capillary bronchitis. In such cases we want to stimulate efficient coughing rather than to suppress it with opiates. In such coughs, however, as are to a great extent irritative or reflex, small doses of opium or morphine, or, still better, of heroin, are most needful to procure rest, and to allay irritation and spasm, and to avoid the distressing sleeplessness and fatigue which such objectless attacks of cough produce. The cough, associated with hæmoptysis, which, unless allayed, will keep up the loss of blood, must, of course, be checked by opiates Indeed, no remedy is more important for the arrest of certain forms of hæmoptysis than small doses of morphine to relieve the cough. It is obvious that the jar and strain of repeated coughing are especially calculated to keep up the bleeding from the lung.

There is one form of asthma, in the management of which I consider opium should be especially avoided—I allude to those attacks of uræmic asthma, which occur not infrequently, in the course of cases of chronic nephritis. I have known so small a dose as half a grain of extract of opium given in such a case induce a profound stupor, from which there was no awakening. A more appropriate remedy for this renal dyspnœa is a few whiffs of chloroform.

In a recent correspondence in a medical journal on the treatment of a "common cold," it was remarkable that there was no reference to the value, for this purpose, of a suitable opiate preparation. I say "suitable" because much depends on the preparation, or rather the *combination*, employed. Whoever has suffered from what is known as an acute *streaming* cold, and has felt the almost immediate relief brought about by a dose of morphine, is not likely to forget it. But this drug must be given with care, in order to avoid any unpleasant after effects. The best combination, in my experience, consists of one-quarter to one-third of a grain of morphine with a dram of spirits of nitrous ether and three drams of solution of acetate of ammonia, and one ounce and a half of camphor water. This should be taken at bedtime, after a very light dinner, at the very onset of the attack. I have repeatedly taken this dose myself, and prescribed it for others, and it is remarkable how, in perhaps a quarter of an hour, the secretions from the nose will cease, the respirations become free, the soreness and aching of the eyes disappear, and a comfortable night's rest be enjoyed, instead of a sleepless night with constant secretion from the nose causing respiratory obstruction. But, although it relieves the initial stage of an acute coryza in this remarkable way, it does not usually "cure" the attack unless taken at the immediate onset, and, if you treat a cold in this way, the patient should be confined to his room for a day or two, otherwise, on exposure to chill, his symptoms will return.

In giving opiates, it is necessary to remember that this drug checks all the secretions but that of the skin, and it will often occur that quite a small dose of morphine will, in many persons, be followed by very light-coloured motions, from the defective secretion of bile; it is often, therefore, advisable to combine some hepatic stimulant with the morphine, such as a few grains of colocynth and blue pill.

When opium is given for the relief of severe pain, the dose has often to be a large one, for intense pain seems to neutralise the neurotic effect of opium to some extent, just as great exhaustion and loss of blood will neutralise the intoxicating effect of alcohol, so that the dose of opium must often be calculated according to the severity of the pain. It is safer, however, to give two successive medium doses than one very large one.

There is great variation in the tolerance of opium in different individuals. Patients, with active secreting organs, and with catarrhal tendencies, generally bear opium well: they are usually of fair complexion, and are rarely the subjects of headache or of "bilious" attacks. But persons, with defective secretions, so-called "bilious" patients, with a tendency to constipation, and to passing pale and offensive motions, and scanty, high-coloured urine, are not, as a rule, good subjects for opium. They are very often persons of dark complexion.

To the foregoing, more or less *general*, remarks on the uses of opium, we may add a brief summary of the various affections, in the treatment of which this drug may be found of value.

It is of great service in many gastro-intestinal diseases.

In *acute gastritis*, it is indispensable for the relief of the pain, when severe.

If the stomach is known to contain irritating substances, it is best administered in the form of hypodermic injection of morphine ($\frac{1}{8}$ to $\frac{1}{4}$ grain), but when we know that the stomach is free from the presence of irritating substances, it is better to give opium by the mouth, so as to bring it into direct contact with the inflamed and irritated mucous membrane. It may then be often advantageously combined with bismuth and hydrocyanic acid, and small doses will generally be adequate ($\frac{1}{4}$ or $\frac{1}{3}$ grain of the extract of opium, or 4 to 6 minims of Battley's solution).

Small doses of opium are likewise serviceable in some aggravated cases of *chronic gastric catarrh*, with much pain and nausea ; in such cases also, it is well to combine it with bismuth and hydrocyanic acid ; or it may be given ($\frac{1}{2}$ grain of the extract) with argentic nitrate ($\frac{1}{4}$ to $\frac{1}{2}$ grain) an hour before food.

Cases of *chronic gastric ulcer* can rarely be satisfactorily treated, without occasional recourse to opium, in order to calm the severe attacks of pain and vomiting which are apt to occur in the course of this disease. Here, again, it may be combined with bismuth and hydrocyanic acid ; or, in *hæmorrhagic* cases, small doses of morphine ($\frac{1}{6}$ to $\frac{1}{4}$ grain) may be given hypodermically, or small enemata may be given, each containing 5 to 10 minims of tincture of opium.

In *perforation* of the stomach in consequence of gastric ulcer, if, for any reason, operation cannot be immediately had recourse to—the patient should be brought quickly under the influence of opium, preferably by hypodermic injections of morphine, or by opiate enemata. Twenty drops of tincture of opium in 2 ozs. of thin starch mucilage may be injected every hour into the rectum, or $\frac{1}{3}$ grain of morphine may be injected hypodermically every hour for three or four doses. The free use of opium will relieve the patient's sufferings, and calm the nervous system, if it does nothing more.

In *cancer* of the *stomach*, opium will be needed in the same way and for the same purpose as in the preceding cases—chiefly to relieve the attacks of severe pains—but its use should be deferred as long as possible, as it aggravates the co-existing constipation, and may take away even the little appetite for food which the patient may have.

I have already referred to the need of much caution in the use of opium in treating *painful* affections of the intestine. In dealing with attacks of enteralgia or colic, the chief indications must be derived from the cause (if discoverable), of the pain. If offending ingesta are the cause, it would be most unwise to give such doses of opium as would tend to arrest the expulsive efforts of the bowel. But as, in many cases, the pain is aggravated by spasm, and no remedy is so efficacious as opium for allaying spasm, in such cases it is useful to combine a small dose of opium with an unirritating aperient, and it is a common practice to give a few drops of tincture of opium with a tablespoonful of castor oil. Or liniment of opium may be rubbed into the abdominal surface with the warm hand, and thus gentle massage can be combined with the other remedies, and this will often be found very useful in the relief of colic depending upon offending ingesta or flatulent distensions. But large doses of opium given internally, suppress peristaltic action, and may even paralyse the muscular coat of the bowel, and although the pain may for the time be relieved, the cause of the colic is not removed. Such cases are often speedily relieved by two or three successive large warm enemata, while an opium or belladonna ointment is rubbed in over the abdomen. In cases in which there is great intolerance of pain, we may begin by giving a hypodermic injection of $\frac{1}{4}$ or $\frac{1}{3}$ grain of morphine with $\frac{1}{100}$ grain of atropine, and then proceed to the use of enemata.

The advisability of using opium in the treatment of diarrhœa must be decided on with caution. In nearly all cases of severe and continued diarrhœa, opium, in small doses, will be needed sooner or later, to allay spasm, soothe the irritated bowel, and allay the general distress and anxiety. But most simple forms of diarrhœa tend to get well of themselves with rest and suitable food, and some cases which have obviously arisen from unsuitable or excessive feeding, are best treated by a mild aperient, and opium is not indicated.

But it would be an error to allow a diarrhœa of this kind, so common in infancy, and early childhood, to run on unchecked, from a reluctance to give a small dose of opium. The dose, however, must be very small, and carefully graduated to the age of the child. Half a grain to a grain of Dover's powder, or a grain to two or three of compound Kino powder conveniently supplies the small doses needed. The effects must be carefully watched in young children, and the dose must not be incautiously repeated.

In treating cases of *acute dysentery*, opium is of great service. The late Dr. Washbourn stated that he had seen lives saved in South Africa by relieving the distressing tenesmus by morphine suppositories. Instead of the morphine suppositories, small enemata may be given, consisting of an ounce of cold starch with 15 minims of tincture of opium. However much some authorities may object to the free use of opium in dysentery, no one can doubt the necessity of allaying the extreme distress of the patient by the local use of opium for the immediate relief to the pain and tenesmus in the rectum.

I have already referred to the need of great caution in the use of opium in cases of intestinal obstruction—in cases of perityphlitis, and appendicitis.

In *acute peritonitis* it is usual, in order to keep the bowels at *rest*, and to relieve pain and vomiting, to give opium freely at starting, a grain of opium every hour until the patient is free from pain. Or ¼ grain of morphine may be given hypodermically, and repeated, if necessary, every two hours, or a small starch enema with 20 minims of laudanum may be administered every hour until relieved.

It will not be advisable to follow this plan in dealing with cases of peritonitis, occurring in the course of chronic Bright's disease, as opium is badly borne by the subjects of that malady. And, in all cases, the opium, it must be remembered, is given to relieve pain and shock, and, as soon as this object is effected, the opium should be discontinued.

Some discussion has arisen as to the advisability of giving opium at all in acute peritonitis, and treatment by saline purgatives has been advocated as more rational, if we recognise the septic origin of this disease, and that the chief danger is the absorption of toxic products. But I am disposed to think that opium, given at the onset, not only relieves pain and distress, but also exerts a direct remedial effect, acting through the vaso-motor nerves on the vessels of the inflamed membrane, just as it does in the nasal mucous membrane in acute coryza, causing their contraction, and so diminishes congestion and lessens exudation ; by checking peristaltic action, it keeps the inflamed parts at rest and, by relieving pain, it quiets the nervous system, and saves the patient's strength. At the same time, we may promote intestinal antisepsis by suitable remedies by the mouth, and by irrigation of the large intestine by suitable antiseptic fluids.

I may again say that the use of opium is not advisable in those obstructive cases, in which hard, impacted fæces may possibly be the cause ; in such cases the sub-paralytic condition of the intestinal walls is aggravated by that drug, and it becomes very difficult afterwards to excite them to efficient contraction. But, in such cases, the peritonitis is usually localised, and the local application of opium and belladonna liniment may be permitted. There is another objection to the use of opium in these cases, and that is, if surgical interference is unavoidable, it leads to needless delay by masking symptoms, and producing a false sense of security, so that precious time is lost, and the patient's chances of recovery greatly diminished.

The combination of calomel with opium, in the treatment of acute peritonitis, as practised by Graves, and many of the past generation of physicians, may not, after all, have been irrational, for the calomel would act as an effective *intestinal antiseptic*, while the opium would relieve pain, allay spasm, and prevent the calomel from exerting an immediate aperient effect, which it might, however, usefully do after the more acute symptoms had subsided, and then its eliminative action might prove most serviceable.

In diseases of the *circulatory* system the use of opium is not frequently indicated. In acute pericarditis and endocarditis of rheumatic origin, with severe præcordial pain, a warm linseed-meal poultice, well sprinkled with laudanum, applied over the heart, may be found useful, or 5 grains of Dover's powder combined with 20 grains of salicine or sodium salicylate and some acetate of ammonia may be given every three hours, for three or four doses. As soon as the severity of the pain is relieved, the opium should be withdrawn.

In chronic valvular lesions, opium must be regarded as a rather dangerous drug, and should always be used with great caution.

In mitral cases with the usual tendency to visceral congestion, it can rarely be safe. I have, however, seen small doses of codeia of great use in some cases of sleeplessness, associated with advanced valvular lesions.

In cases of aortic incompetence with nocturnal dyspnœa and sleeplessness, it is sometimes needful to have recourse to opium or morphine for the relief of these symptoms, and, to avoid any remote depressing effects of the drug, we should combine it with ammonia and ether, and use as small doses as possible consistently with the relief of the patient. Some French authors maintain that, in these cases, morphine increases the amount of blood circulating in the brain, and so relieves the cerebral anæmia.

But the good effect of opium in these cases is mainly due to its regulating and sedative action on the excited cardiac muscle. It steadies the heart, enables it to do its work more effectively, and it quiets the nervous disturbance. I prefer giving it by the mouth, as safer than by hypodermic injections ; $\frac{1}{8}$ to $\frac{1}{4}$ of a grain of morphine combined with 30 minims of sal volatile and 20 minims of spirits of ether in an ounce of peppermint water may be prescribed at bedtime. Much the same course may be followed in treating attacks of angina pectoris when the pain is very severe and is not relieved by the nitrites.

In such severe cases, we may use morphine ($\frac{1}{4}$ to $\frac{1}{3}$ gr.) hypodermically combined with strychnine ($\frac{1}{40}$ to $\frac{1}{20}$ gr.).

So also in the course of aortic aneurism, we may find it absolutely necessary to use morphine in order to allay its distressing symptoms. Hypodermic injections of morphine and atropine and the local application of opium and belladonna liniment will be frequently required.

Some of the symptoms associated with *respiratory* diseases, as I have already pointed out, call for the cautious administration of opium for their relief. Heroin is, I think, better suited than other opium derivatives for this purpose in many cases. In acute laryngeal catarrh, especially that form so often connected with influenza, with scanty tenacious secretion difficult of expulsion, morphine sometimes seems to aggravate the scantiness and tenacity of the mucus, unless it is combined with minute doses of tartarised antimony, or apomorphia, but the latter drug has seemed to me to exert a depressing effect on the heart, and, therefore to be unsuitable in influenza cases.

Heroin, however, appears to be quite as efficacious in quieting the cough as morphine, and to have the advantage of loosening the tenacious mucus.

Whatever opiate we use to allay the cough of acute affections of the upper air-passages should be associated with alkaline expectorants such as bicarbonate and chloride of sodium, or ammonium, or such alkaline mineral waters as Ems, or Bourboule, or Apollinaris. Codeia, in small doses, also answers well in many of these cases.

It is necessary to be extremely cautious in the use of opiates, in cases of *diffused acute bronchial catarrh*, because of the risk of checking expectoration by diminishing the sensitiveness of the bronchial mucous membrane, whereas our obvious duty is to facilitate and promote expectoration by all means at our command. It is very hazardous also to give opiates to relieve the cough in cases of bronchial catarrh in the aged and feeble, but I have already mentioned this point.

At the onset of *acute pleuro-pneumonia* with severe pain in the side on inspiration, an opiate is often indicated, such as 10 grains of Dover's powder with a few drams of solution of acetate of ammonium at night, and, locally, a hot linseed poultice sprinkled well with laudanum. The same indication occurs in the early stage of acute pleurisy.

In the treatment of *advanced phthisis*, and the serious symptoms which are apt to occur late in this disease, opiates are indispensable. The cough is often most distressing, especially when the larynx is affected, and we must all agree with the remark of Pidoux, that "it is impossible to possess too many means for repressing a symptom which so exasperates the disease," or that of another eminent physician, who said, "Without opium the treatment of phthisis would be impossible!" Keeping in mind what has been already said as to the need of great caution and discrimination in the method of giving opium—that its object is to allay irritation, and not to suppress expectoration—and limiting our doses as much as possible short of sacrificing efficiency, we must do all we can to help the patient against the exhaustion which this distressing symptom produces.

Heroin and codein are the two best derivatives of opium for this purpose. A combination of opium and belladonna often answers well, especially where night-sweats are brought on by coughing. When the cough is attended by *hæmoptysis* it is extremely important to allay it by opiates; it not only prevents the jarring movements of the chest, which tend to maintain and provoke this symptom, but it soothes the patient's nervous system, quiets the circulation, and lowers blood pressure.

When troublesome *diarrhœa* is added to the other symptoms, we have an additional indication for the use of opium. A good method is to give 5 to 10 grains of Dover's powder, or 10 to 20 grains of compound Kino powder, and 10 grains of tannin, mixed with 2 ounces of starch mucilage as an enema, injected into the rectum twice daily.

In the treatment of *acute rheumatism*, before the introduction of the salicylates, opium was found very serviceable, in the early stage, in assuaging the pain, lessening the cardiac excitement, and quieting the restlessness. There are cases in which it still may be found useful. It is best given in the form of Dover's powder, 10 to 15 grains, with a little acetate of ammonia, in a draught at night.

In the painful joint conditions, and in the neuralgias, and myalgias associated with chronic rheumatism and gout, it is rarely desirable to have recourse to opiates, except as an occasional and temporary expedient for the relief of very severe pain; the objection to its use is that it lessens the activity of the excreting organs, which it is very undesirable to do, especially in gouty cases. There can, however, be no objection to its external use in the form of lotion, liniment, or plaster, and, combined with belladonna, it often gives great relief. In the various forms of neuralgia and neuritis, a hypodermic injection of morphine is often needed to relieve the severe pain and procure sleep; but it is important to exercise great caution in its use, for it is precisely in these cases, which are commonly met with in neurotic subjects, that the risk of establishing the morphia-habit is to be feared.

There is a general consent as to the value of opiates in diabetes. Some prefer morphine, others codein. Seegen found morphine and Carlsbad water the two most efficacious remedies in diabetes, and he testified that he had found morphine diminish the amount of sugar excreted in the urine in both the slight and the severe forms of this disease. We should begin with small doses—morphine, ⅙ grain thrice daily, which may be increased slowly to 1 grain, or codein ½ grain, similarly increased to 4 or 5 grains three times a day. Diabetic patients tolerate large doses of opium remarkably well, and large doses are often needed in order to produce any decided effect.

The foregoing observations on the use of opium are the results of long clinical experience, and may, perhaps, prove of use to younger practitioners in applying, to the relief of sufferers, the drug that has been fitly named "God's gift to man."

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THE INDICATIONS FOR THE USE OF OPIUM IN ACUTE DISEASE.

By **RALPH STOCKMAN, M.D.,**
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In discussing the indications for the therapeutical use of opium in disease, it is always to be borne in mind that treatment by it is symptomatic and palliative, rather than directly curative. It does not strike at the cause of any disease, but nevertheless its indirect effects, as a remedial agent, may be of prime importance in giving to the patient the benefits of sleep, freedom from pain, and rest of body and mind—factors which are often of the utmost value in determining the favourable issue of an illness. In these respects it is unrivalled, for, unlike most of our drugs, no substance has ever been discovered which makes an efficient substitute for it. The action of opium is determined by the action of morphine, its principal alkaloid, and, in practice, the latter very largely replaces it. Opium preparations, however, are still preferred for many purposes, and especially when it is desired to act upon the alimentary canal, as its local effect is more prolonged and gentle than that of morphine, which is more quickly absorbed. In man, the predominant action of opium and morphine is depressant, and is exerted specially on the higher and sensory parts of the brain, and on certain of the medullary centres and visceral ganglia, the spinal cord being scarcely affected. In addition, it diminishes the secretions of the alimentary canal, and also, through the nerve-ganglia, lessens the activity of its muscular fibres, a similar action being exerted on the other non-striped muscles. The indications for its use in acute disease depend directly on these pharmacological actions. Speaking broadly, it is employed chiefly to abolish pain and sleeplessness, to lessen spasm of involuntary muscles, to abate certain forms of dyspnœa, and to diminish secretion from the alimentary and bronchial mucous membranes. When we come to consider the particular acute conditions in which these indications render opiates valuable or necessary, it is impossible, in the limits of a short paper, to do more than glance at the most commonly occurring of them.

In acute pneumonia, if the pain of an accompanying pleurisy is not banished by an ice-bag or poultice, morphine may be given hypodermically with almost certain success, as it not only acts on the sensorium, but also tends to diminish the frequency and depth of the respiratory movements by an action on the centre. Only one administration is usually required, as effusion soon supervenes, or the lung becomes solidified by exudation, and, in either event, friction between the two pleural surfaces ceases, and with it the pain. Later on in pneumonia, if the patient is sleepless and excited, the ordinary hypnotics are frequently of little value in procuring a good night's rest, and then also morphine may be given. Fears are sometimes expressed that, in these circumstances, it is too depressing for the heart and circulation, but, in reasonable dose, opium and morphine exercise no depressant cardiac action, and experience has abundantly shown that they are safe and valuable hypnotics in pneumonia. The only contra-indication is when the lungs are so consolidated by exudation, or so embarrassed by bronchitis, that a sufficient oxygenation of the blood cannot take place, in which circumstances, it is inadvisable to further lessen respiratory activity by diminishing the sensitiveness of the centre with morphine. In the sleeplessness of enteric and other acute fevers, opium may be given, provided that there is no marked œdema of the lungs, or nephritis. Recently, I have used morphine with advantage in cases of cerebro-spinal meningitis, in which pain, cortical irritation, and excitement were present.

In the shock, severe pain, and psychical disturbance, which follow on such accidents, as burns, severe injuries, and corrosive poisoning, morphine mitigates the inevitable suffering, and the nervous excitement which is apt to accompany it. In hæmoptysis, hæmatemesis, and hæmorrhage into the bowel, it has a similar sedative effect, but, further, by the quietude which it induces, the formation of a clot is rendered more likely, and it thus assists in stopping the hæmorrhage. The hypodermic injection of morphine sometimes causes nausea and even vomiting, owing to its excretion from the blood through the stomach mucosa, and this may sometimes prove a drawback to its use in hæmatemesis, but the disturbance is very temporary, and is usually succeeded by a long period of quiet.

In acute peritonitis, opium, or morphine, not only lessens the severe pain and gives the patient much-needed sleep, but it gives rest to the inflamed tissues by lessening peristalsis. In perforation, its use has been objected to on the ground that it masks the symptoms and gives a false feeling of security, because the patient under its influence seems to be better than he really is. But, in all cases, operation should be performed at once, and hence it does not matter whether opium masks the symptoms or not. In acute appendicitis, the same objection has been made to the administration of opiates, but the pain and discomfort complained of by the patient should not form the criteria determining operation, the necessity for which should be decided by the local signs, the leucocytosis, temperature, pulse, and general condition.

The irritation and coughing from acute pharyngitis, laryngitis, tracheitis, and bronchitis, in cases in which there is scanty secretion, can be abolished by small doses of opiates; but if there is much secretion, it is best to avoid giving opium, and to encourage the expectoration of the mucus.

The value of morphine in lessening spasm of involuntary muscles is seen in the treatment of lead colic, where the action of the metal induces a painful contraction of parts of the bowel with severe constipation. The relaxation of the spasm allows purgatives to act satisfactorily, and abolishes the severe pain. In tenesmus from any cause, a suppository or enema containing opium is usually efficacious. In hepatic and renal colic, it is our most valuable remedy, and here, as in lead colic, it may be advantageously combined with belladonna, or nitrites. In strangulated hernia, it may aid reduction, and in severe vomiting, obstruction of the bowel with spasm, spasm of the cervix uteri, irritation of the bladder, and similar conditions, its anti-spasmodic action may often be used with great benefit. In all such cases, morphine probably acts by depressing the local nerve-ganglia which control the muscle fibres.

In treating dyspnœa with morphine, great discrimination in its use has to be observed. In pulmonary dyspnœa, as in pneumonia with much consolidation, or bronchitis with excessive secretion, in œdema of the lungs, and in general dropsy from cardiac or renal disease, morphine should be regarded as dangerous, owing to its depressing effect on the respiratory centre, which should be maintained in as active and as sensitive a state as possible. But, on the other hand, when dyspnœa is a prominent symptom in aortic disease, dilated heart, and pericarditis, and in attacks of so-called cardiac asthma, and when the respiratory disturbance is primarily of cardiac origin, and the activity of the centre is exhausting and detrimental to the patient rather than beneficial, morphine should be given. The respiratory centre, in these cases, is probably unduly excited, and the drug exercises a calming effect on it. In the distress of a sudden pneumothorax, it often gives great relief in the same way. Many cases clinically present a mixture of these two causes of dyspnœa, and then much care requires to be exercised in the use of opiates. In bronchial asthma, nothing gives such complete and lasting relief as a hypodermic injection of morphine, but its action here is really an anti-spasmodic one, and comparable with that on other non-striped muscles. Some time ago, Dr. Thomson, of Uddingston, drew my attention to the fact that $\frac{1}{12}$ to $\frac{1}{8}$ grain of morphine, given hypodermically, with 5 to 8 minims of adrenalin solution (1 in 1,000) was even more active than morphine alone in cutting short the spasm, and this I can confirm, from my own experience of several severe cases.

It is hardly necessary to dwell on the usefulness of opium in checking acute diarrhœa from any cause, or, on the necessity, in certain cases, of first giving a purgative. In bronchitis, with much watery secretion, small doses, combined with belladonna, sometimes greatly help in checking the discharge.

In motor disturbances, such as are seen in chorea, tetanus, strychnine poisoning, and pressure on the cord, morphine is of no value except to deaden pain or procure sleep, as it has very little action on the spinal cord, and in such conditions, chloral hydrate and bromides are much more active. In bulimia, in gastralgia, in hiccough, and in many other conditions, morphine is constantly being employed, but its numerous applications can be included under the general indications previously outlined, and need not be further particularised in detail.

CHRONIC MORPHINISM, WITH SPECIAL REFERENCE TO TREATMENT.

By **W. E. DIXON, M.A., M.D.,**
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THOSE who indulge in the habit of taking morphine may be divided into three classes, the opium eater, the opium smoker, and the morphinist who injects the alkaloid under the skin. Of the opium eater we need say little as the practice is not common in Europe, but everyone, who has read the evidence given before the Opium Commission in India, must be aware that the practice of opium eating in the East is not necessarily a vicious indulgence. The drug is taken in the belief that it is an antidote to fevers and certain other diseases, and is resorted to in much the same way as we resort to caffeine, or tobacco. In the fen districts of England, the tradition of taking opium to ward off malaria still exists.

Opium smoking is also extremely rare in this country, although it is less uncommon in the United States, and is especially prevalent amongst the Chinese. The amount of morphine absorbed into the system as the result of smoking is very small, first, because, relatively, very little opium is smoked, and, secondly, because a considerable proportion of its alkaloid is destroyed by the heat. But the morphine taken in this way by inhalation is absorbed with extraordinary rapidity, and produces its effects immediately. At first, there is a sense of elation which is quickly followed by depression, and sleep, the whole effect not lasting longer, probably, than three or four hours. Smoking cannot be regarded as so injurious as either opium eating or injecting morphine. In Eastern countries it is reputed to enable men to do more work with less fatigue and on little food ; but the smoker becomes a slave of the habit. The vice, at all events in the West, is apt to be indulged in the worst possible surroundings, and this adds to the evil.

Morphine injection is the worst form of the habit, and, unfortunately, is the one most prevalent in this country. It is rarely difficult to trace the origin of the practice. There are those who have learnt to inject the drug for the relief of pain during the progress of a fatal malady. In these, as in normal people, the dose must be ever increased to produce the desired effect. There are others, in whom the habit is formed by a few injections given for the relief of chronic neuralgias, and the like ; and, in this connection, it cannot be stated too forcibly that, under no conditions, should patients be entrusted with the morphine syringe by their medical attendant. Doctors, chemists, and others, who have the handling of drugs, are especially liable to contract the habit. By far the greater number of habitués to morphine are those of whom we speak as neurotics. We are all reflex in our thoughts and actions, and each sensation after perception induces a train of thought, or arouses an action, but the response, whatever nature it may assume, is influenced by the highest or controlling centres, and is not a simple reflex from lower centres. The neurotic may be regarded as one whose perceptions of sensations are hyper-sensitive ; sensations are not appreciated in proportion to their significance, and, therefore, what we may regard as lower reflexes are increased. Whether this is due to hyperactivity of the lower centres, or to want of controlling power in the higher, need not here detain us ; the fact remains that it is such people who become slaves to drug habits.

The morphinomaniac must continually increase the dose of alkaloid in order to get the required effect. The explanation of this acquired tolerance, which applies not only to morphine but also to other drugs to which tolerance can be attained, is found in the fact that the tissues acquire the property of oxidising and completely destroying the drug. Morphine is excreted by the alimentary canal, and from the fæces of the normal animal about 70 per cent. of the morphine previously given by the mouth, or subcutaneously, can be extracted. But, if the dose is continually administered, the morphine in the fæces dwindles to the vanishing point, and the reason such enormous doses of morphine can be taken into the system (as much as 60 or 70 grains of morphine have been injected in 24 hours) is that the larger part of the alkaloid, according to the degree of acquired tolerance, is destroyed in much the same way as starch or alcohol.

The symptoms of morphinism, at least in the earlier stages of the habit, are rather moral than physical, but, later, when the habit has fully developed, the victim loses flesh, assumes a sallow, haggard, and aged appearance. His mouth becomes parched, and there is commonly gastric catarrh in a greater or less degree, and chronic constipation.

The one object to be accomplished by the treatment is to withdraw the morphine, with the least possible inconvenience to the patient, and to render the craving bearable. In order to do this effectually, some form of retreat or seclusion is almost a necessity. Home treatment is practically impossible; it is difficult for a relative to stand firm against the terrible cravings or neuralgias of the patient, and friends can rarely successfully penetrate or defeat the wiliness and deceit of the victim. No medical man can treat under such conditions, and a tractable patient is almost certainly getting the drug on the sly, for what is so especially discouraging to him is the certainty that, if once the craving is felt, it will cause increasing distress until the morphine is given. The morphine habitué may give up the habit himself; but, after he has consented or even begged to be placed under restraint, and if his request is granted, he will do all in his power to defeat its object. Not infrequently he will even pretend that he is cured rather than confess to his doctor that he has cheated. Moreover, it must be remembered that these patients never present themselves for treatment until it has been shown, by many trials, that they cannot cure themselves.

How can we account for the cravings of the morphinomaniac? Morphine depresses sensory nerve cells in the reverse order of their development, and the dreams, the brilliant imagination, and the general feeling of exhilaration experienced by the habitué after an injection, are caused by depression of the highest and controlling centres, and are but the expression of hyperactivity of the lower ones. At no time is there any evidence of increased mental activity involving greater control, judgment, or attention, faculties which are formed late in development. Later, the lower centres also are affected, and sleep ensues. As the action passes off the sensory nerve cells, after their prolonged rest, become hyperactive in the same way as our senses are hyperactive after sleep. In the lower animals, in which the cerebrum has not developed, morphine may cause typical strychnine-like convulsions when given in extremely large doses; but, whilst strychnine acts mainly upon the sensory cells of the cord, morphine acts mainly on those of the cerebrum. If the cerebrum should be deficient or absent in an animal, very large doses of morphine will still affect the cord. When the morphinomaniac passes from the influence of the drug, his psychical centres become hypersensitive, so that the interpretation of sensations is exaggerated out of proportion to their significance. The patient is at the beck and call of every stimulus, and his existence becomes intolerable. His blood pressure rises, and respiration is increased, in both cases the result of excessive medullary stimulation. The most serious danger which may follow sudden withdrawal is collapse. In this case, when the craving comes on, the patient becomes more and more excited, he is continually moving about, and may suffer from tremor and rigors. The skin becomes covered with sweat, but the face is pale, he salivates freely, his pupils tend to dilate, and vomiting and diarrhœa are not uncommon. Not infrequently hallucinations of vision, smell, and hearing occur which may be succeeded by violent delirium. It is at this stage that collapse may ensue; the pulse gradually ceases to be felt at the wrist, and the signs of consciousness fail. This condition may, of course, be relieved by the injection of a little morphine, but as the sudden withdrawal of morphine is now no longer practised, it is rarely seen. The cause of this collapse is the same as that which may arise from any sufficiently severe peripheral stimulation. Cerebral activity, especially in the lower centres, is inhibited so that respiration becomes shallow, and the blood collects in the enormously dilated splanchnic vessels.

Sudden withdrawal is not only dangerous to the patient, but it involves very severe suffering, from which recovery is gradual. Treatment by sudden withdrawal, except in those mild cases in which only two or three grains are consumed in the 24 hours, should be abandoned. Assuming that our patient is in some institution or home, we may at once start cutting down the use of the drug. In patients shattered in health, suffering from cachexia, and emaciation, treatment should be as brisk as possible. For those, who are in the habit of taking very large doses, begin the cutting down process by diminishing the morphine in the first 24 hours to one half the usual quantity. This may be reduced again and again until, in 8 or 10 days, the dose should reach the vanishing point. It is the last 2 or 3 grains which present the serious difficulty, and it is then that the patient, who may have been docile during the earlier treatment, is apt to rebel. So that at this time relapses are especially common. It is sometimes advisable at this stage, according to Dr. Jennings, to substitute opium per rectum for morphine injections. The advantages of rectal injections are that the drug is absorbed more slowly, and so the "stimulation" stage of the hypodermic injection is almost absent; moreover, alimentary troubles are largely avoided. At this stage, the astute morphinist will employ all his cunning to obtain his fetich, and success now largely depends on the integrity of the attendants. Everything reaching the patient must be carefully examined, and all untoward symptoms, such as sleeplessness and diarrhœa, must be determined by the nurse's record.

Collapse, violent diarrhœa, and excessive excitement should be avoided by this method of treatment, whilst the patient's sufferings are not unduly prolonged; the symptoms and excitement may be so pronounced, however, as to warrant the use of some cerebral depressant. Chloral, sulphonal, codeine, paraldehyde, cannabis indica, hyoscine, the bromides, and the synthetic morphine derivatives have all been employed for this purpose. The morphine derivatives should, however, be avoided, as they lead to a habit no better than that which we are trying to destroy; whilst sulphonal, on account of its tendency to cause sudden coma and hæmatoporphyrin in the urine, is a drug at all times better dispensed with.

Bromides are also useful to calm the excitement, but they do not act in the same way as morphine, since the latter drug acts on sensory nerve-cells alone, whilst the former depresses all nerve-cells. Cocaine is at all times a most mischievous agent, and even as a temporary substitute is better avoided.

Atropine and its allies are the drugs, which have been put forward to such a large extent as "cures" for the various drug habits. Hyoscine is by some authorities strongly advocated for the morphine habit ; the patient is kept under its influence for two or three days, during which time the morphine is withdrawn. Hyoscine, besides having all the typical actions of atropine on glands and muscle, also depresses the cerebral hemispheres, and its value, no doubt, lies largely in the fact of this depressant action on the central nervous system. Atropine and mandragorine, which act alike, have also been employed, but with less favourable results. Hyperexcitability is the cause of the craving, and it is difficult to see how this can be affected, save by some drug directly depressing the brain. Hence, we can dismiss such drugs as camphor and pilocarpine, which have, nevertheless, been recommended. Dr. Jennings, as previously mentioned, recommends the substitution of rectal injections for the hypodermic when the patient is feeling the withdrawal of the drug. He also prescribes Turkish or hot-air baths, alkalies, and cardiac tonics. Alkalies are certainly very useful, as the morphinist is especially likely to suffer from stomach troubles and hyperacidity. The value of cardiac tonics is more doubtful, first, because morphine has no action on the heart, depressant or otherwise, and, secondly, because the morphinist during the process of withdrawal has a very high blood pressure, and this should, if necessary, be combated rather by vaso-dilators than by digitalis and the like.

Syncope and collapse are rarely seen since sudden withdrawal has been given up ; they are treated in the same way as collapse from other causes, by the use of warmth, the injection of caffeine, and possibly, in emergencies, by the intravenous injection of squill, or adrenalin.

A generous diet is of the utmost importance, and everything possible should be done to assist absorption. This is frequently deficient, because gastric catarrh is almost invariably present, not uncommonly accompanied by nausea and vomiting. Alcohol, if possible, should be used sparingly, but if the tissues are seriously depleted, and food is taken badly, then some kind of alcohol is a necessity, since we know that this body is always very rapidly absorbed, and, by oxidation, provides the body with energy. As nutrition improves, massage and tonic douches may be tried. The patient should be kept as much as possible in the fresh air, and later, should become accustomed to mild bodily exercise. Sometimes the pains, which were the origin of the habit, return with the suppression of it, but as nutritious food is pressed upon the patient, and the body weight is regained, these tend to disappear.

Morphinists show a strong disposition to relapse, and, not infrequently, the reclaimed morphinist turns to alcohol. Levinstein records 61 relapses out of 82 patients, and Oppenheim quotes 93 relapses out of 250 patients. These figures are, however, probably too high. They include alcoholics and semi-insane people of all sorts. Assuming that the withdrawal has been completed in a fortnight, we have next to determine for how long the patient should be kept in seclusion. All observers are agreed that the patient should not return home, if possible, until after about the lapse of a year. He may well be kept in seclusion for, say, two or three months, and then sent for a sea voyage, the medical man on board being duly informed as to his former habits. A travelling companion, possessing some influence, is also desirable, and he should understand the importance of preventing his charge from obtaining narcotics or alcohol.

In severe cases, dyspepsia, melancholia, general weariness, loss of appetite, and diarrhœa may pursue the patient on and off for a year or two after the cure. These fluctuations in health are accompanied by the intolerable yearning for relief which is satiated by narcotics. If the patient has judicious friends, and is carefully watched, these tides of temptation are withstood; but the patient, who has been turned out at large and "cured" after three or six months' residence at an institution, is very liable to relapse.

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HYPERTROPHY OF THE PYLORUS IN ADULTS AND ITS TREATMENT BY OPIUM.

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Hypertrophy of the muscular tissue of the pylorus is found under the following conditions :—i. As a complication of disease near the pylorus, such as gastric, or duodenal ulcer, and carcinoma. ii. As a complication of movable kidney. iii. As the result of a gastric motor neurosis. iv. As the so-called congenital hypertrophic stenosis of the pylorus in infants.

It is also found in association with a gastritis, but whether the gastritis is ever the primary condition is doubtful, although there is nothing inherently improbable in such a causal relationship. Pathologically, there is a thickening of all the layers of the pylorus, but the most striking condition is the hypertrophy of the muscular layer, which is due to a frequently recurring overaction or spasm of the muscle.

Spasm of the pylorus is a common occurrence, of local origin in acute forms of dyspepsia from various causes, especially in children, or it may occur as the result of some influence from the central nervous system, as in certain emotional states; but, in order to produce hypertrophy, the spasm or overaction, from whatever cause, must be frequently repeated for some weeks. The association of this condition with ulcer and carcinoma is well known, and is to be regarded as a complication of these diseases, whereas the motor-neurotic form, if it may be so termed, has had less attention paid to it, and better lends itself to separate consideration; hence it will be the form principally dealt with here.

The following case may fairly be taken as typical of the motor-neurotic form.

A female, aged 54, in good circumstances. With the possible exception of a strong tendency to phthisis, the family history does not appear to be important. Always very strong, six children. In November 1904, she had great anxiety and worry, became nervous and run down. After a long and cold drive, and entertaining friends in the evening, she was suddenly seized with epigastric pain after food. Pain recurred next day, and soon began to follow each meal. Constipation became troublesome. Pain came on about a quarter of an hour after meals, sooner, if the patient did not rest. Sometimes the pain was accompanied by regurgitation of food, and flatus became troublesome. Movements were now felt "high up on the left side" of the abdomen, and, shortly after this, vomiting would occur, followed by relief, so that the patient could go about as usual fairly free from pain. No loss of appetite throughout. Tea seemed, at first, to cause most pain, and, when no fluid was taken with the meals, the pain was less. At this time I gather that bismuth and soda were taken, but with no benefit, and soon every meal was vomited. When I saw her, in January 1905, the stomach would not retain a glass of water, and the vomiting had increased in force. There was loss of flesh, no obvious anæmia, lungs and heart normal, except that the pulse-rate was easily raised. Abdominal walls tender, and said to be thinner than formerly. A succussion splash easily obtained; greater curvature of stomach, did not reach downwards quite to the umbilicus. A tender swelling, the size of a walnut, moving downwards on inspiration, was felt just to the right of the middle line 2 inches below the tip of the xiphoid cartilage. The history of worry, the absence of previous dyspepsia, the sudden onset, the early appearance of sensible gastric movements, considered together, suggested a nervous origin. The patient was removed from home, relieved of all responsibilities, and kept under daily observation. Examination of the vomit showed presence of free hydrochloric acid. The patient was given small quantities of milk frequently repeated, sometimes varied with raw egg and lemon-juice, and a mixture of bromides with valerian and sumbul taken. Some slight improvement followed, but, although lessened, the vomiting continued, and the pylorus was unchanged. A week later, 5 minims of tincture of opium was added to each dose of the mixture unknown to the patient. The effect was remarkable, the vomiting practically ceased, the pain was much lessened, and the pylorus, in a few days, was not felt, nor was this region tender. In a fortnight, the patient was taking a full diet, and the bowels were much less confined. In February 1907, there has been no recurrence of the trouble

The second case is one associated with movable kidney.

Female, single, aged 27, domestic servant. Neurotic disposition. One child and one abortion. At work from seven in the morning until midnight, and often called up at night. Sent to Bristol General Hospital, under my colleague, Mr. Lansdown, for fixation of movable kidneys. The patient was unwilling to leave her work, and an attempt was made to retain the kidneys in position with a belt, the patient coming under my care for the neurasthenia. The upper end of the right kidney was a hand's breadth below the costal margin, and the left a thumb's breadth. There was some general improvement for a few weeks, then pain began to occur some 15 minutes after meals. Later, the pain increased until vomiting occurred, which gave relief. In a month's time, although the appetite was good, she vomited everything taken by the mouth. Mr. Lansdown again examined her with me. The abdominal walls were thin, and both kidneys very sensitive. During the examination, the patient became very restless, when suddenly the stomach was seen to bulge forward, and waves of peristalsis, arising under the left costal margin, passed slowly across towards a point where we could feel the pylorus as a tender lump. There had been no sign of air-swallowing, but a quantity of gas was eructated, and the patient made great efforts not to vomit. This attack was said to be similar to her ordinary attacks. 10 minims of the tincture of opium were given three times a day in ½ oz. of water.

In a few days, all vomiting had ceased and ordinary food was taken. Later, Mr. Lansdown fixed the right kidney by operation, the patient doing well. When, however, the question of fixing the left kidney was broached to the patient, the pain and vomiting after meals recommenced. Opium was again given, and controlled the vomiting at once, and there had been no further trouble a year later.

This spasm and hypertrophy, occurring in association with movable kidney, are of great interest. I remember, as a hospital resident, seeing a similar gastric storm in a case of movable kidneys, associated with dilated stomach and an occasionally palpable pyloric tumour.

A female, about 40, had been admitted with a diagnosis of dilated stomach, due to pyloric obstruction, probably malignant. During an examination, at which both kidneys were found freely movable and tender, there was an attack of enormously exaggerated and visible gastric peristalsis with great pain and discomfort. The kidneys were easily kept in place with a belt and pads, and the gastric condition gradually subsided.

In one or two cases, recently under my care, in which epigastric pain, occasionally followed by vomiting, has been associated with movable kidney, the gastric condition has been entirely relieved by opium. It is a difficult question to decide whether the pyloric spasm is caused by the neurasthenic condition, so frequently associated with movable kidney, removing some central inhibitory influence over the pylorus, or by its stimulation, or, on the other hand, by an increased excitability of the pyloric reflex, either due to the hyperæsthetic condition of the kidney, or from the kidney dragging upon branches of the solar plexus. Possibly more than one of these may at times be responsible.

It would certainly appear more probable that a reflex spasm of the pylorus, with resulting stenosis, should be the link in the well-known association of dilated stomach with movable kidney, rather than that the kidney, by pressing against the pylorus, or by dragging upon the peritoneal attachments, narrows the pyloric orifice, as has sometimes been stated to be the case.

It is not necessary to the diagnosis of pyloric hypertrophy, that exaggerated gastric peristalsis should be visible, the abdominal walls may be too well covered, or the peristalsis insufficiently developed, but it would be more frequently observed, were it to be looked for at the time when the pain is at its height. In the following case, peristalsis was not seen.

A middle-aged man was sent to Mr. Lansdown at the Bristol General Hospital with a diagnosis of pyloric carcinoma. Before seeing the case, I examined the gastric contents by Toepfel's method, after a test meal, and found no free hydrochloric acid. In spite of this report, and the presence of a well-defined pyloric tumour, my colleague diagnosed hypertrophy of the pylorus without malignant disease, relying upon the previous history and the course of the disease, there having been a history of worry, a sudden onset, and great variation in the symptoms, which had extended over a long period. He prescribed tincture of opium in 10–minim doses, twenty minutes before each meal. The patient ceased to vomit, the pylorus rapidly became impalpable, and there was a good recovery. At no time were gastric movements seen.

The association of gastritis with pyloric hypertrophy is of considerable interest, for it is difficult to say, in some cases, whether the gastritis is secondary to pyloric obstruction or not. Amongst three cases with gastritis reported by Boas[1] is one associated with ptosis of liver, kidneys, and stomach, in which there are certain features which suggest a neurotic origin. In this case, two days before an operation was to take place, there was sudden improvement, which lasted for five months. On the return of symptoms, a gastro-enterostomy temporarily relieved all symptoms. In spite of the gastro-intestinal communication, the trouble recurred after four months, to suddenly disappear again five months afterwards. Indeed, in all these cases, he states that the pyloric condition preceded, and so much overshadowed the gastritis, that he therefore gives the name gastritis a secondary position in the title of his paper—"Ueber Hypertrophische Pylorusstenose (Stenosierende Gastritis) und deren Behandlung."

Symptoms of the Motor-neurotic Form.—Pain in the epigastrium is the most constant symptom, varies greatly in intensity, is gnawing or twisting in character, increased by taking food, and relieved by vomiting. Vomiting is always present at some time, and tends to become more forcible. Vomitus consists of food or mucus; gastric contents after test meal may or may not contain free hydrochloric acid. Appetite remains good. Constipation is usual. May be considerable wasting. If abdomen examined during height of pain, exaggerated gastric peristalsis may be visible; visible peristalsis is occasionally stimulated by abdominal palpation, and produced or stopped by emotional states, such as fear. Succussion splash is usual. Signs of pyloric obstruction, such as dilatation of the stomach, are frequently seen, and may vary considerably in the same case. Enlargement and tenderness of the pylorus may often be made out, especially if examined for during visible gastric peristalsis. Sudden exacerbations, or amelioration of the symptoms, is very characteristic. Hæmatemesis may occur without presence of carcinoma or ulcer. Condition, if unrelieved, may last for months or even years. That a fatal termination is rare is, I believe, the reason why this condition is so little recognised.

There is often a definite history of worry about the time of onset.

Whilst bearing in mind that carcinoma and ulcer near the pylorus may be present with hypertrophy of the pylorus, the previous history, the suddenness of the onset, and the early appearance of pain after meals, taken together, help to exclude them. Violent gastric peristalsis would be against carcinoma, as the presence of carcinoma tends to produce progressive weakness of the gastric muscle. Unfortunately the examination of the gastric contents is of little or no assistance in excluding carcinoma and ulcer, for Boas[1] has shown, in his cases with gastritis, that hydrochloric acid may be absent, lactic acid and Boas-Oppler bacilli present. Einhorn has shown that absence of hydrochloric acid may occur as a secretory neurosis, and the secretion of mucus may also be reflexly excited, so that these may be associated with the motor disturbance in a neurotic condition apart from a gastritis.

Treatment.—It is a marked feature of the disease that the usual dietetic, and, with the exception of opium, the medicinal treatment, of this motor neurosis yields but disappointing results. I have here no personal experience of lavage, but, judging from its temporarily, and sometimes permanently, good results in the congenital form, one would expect some benefit to be derived from it, but it is not a procedure which commends itself to one in a condition in which neurasthenia is so often a pronounced feature.

In two of the cases given above, there is little doubt that, but for the use of opium, gastro-enterostomy would have been needed for their relief. But operation in this disease appears to me to be peculiarly objectionable. In the one of Boas' cases, to which I have referred as possibly being neurotic in origin, the symptoms returned after a temporary relief by gastro-enterostomy, leaving the selection of further methods of surgical assistance somewhat difficult. But a grave objection lies in the fact that some of these cases die suddenly shortly after operation. Two cases have impressed this upon me. The first was seen in 1897.

A middle aged woman had been treated for years for recurrent gastric ulcer. There had been hæmatemesis on more than one occasion. Whilst in hospital, a severe attack of pain was diagnosed as being due to perforation of an ulcer. During the operation, whilst the stomach was being handled in the search for an ulcer, the organ suddenly became tense and globular, and bulged into the laparotomy wound. It was translucent under the strong artificial light, and a deep groove, due to contracting muscular fibres, slowly travelled along the organ from left to right. The exploration only lasted a few minutes, but the patient died suddenly a few hours later. At the post-mortem, nothing was found to account for death; there was no no trace of past or present ulcer; the only gastric abnormality being a "thickened" pylorus.

For permission to give an abstract of the second case, I am indebted to Mr. Lansdown :—

A man, aged 54, in the Bristol General Hospital in 1904, subject to stomach attacks all his life. Twelve years ago, after hurriedly drinking some milk in lieu of breakfast, he travelled to attend a funeral in London. At the funeral, he had a sudden attack of intense epigastric pain: was unable to work for three weeks subsequently, and sometimes vomited. Two years later he had a similar attack. In October 1903, the trouble recurred. Vomited sometimes every day, and sometimes only at a week's interval Vomit consisted of "churned food or phlegm." Always constipated. On examination, there was found marked ptosis of the stomach; succussion splash, a tender swelling felt over the aorta, passing upwards to the pylorus. Pylorectomy was performed. Three days afterwards the patient died suddenly. At the post-mortem, there was no peritonitis, and the gastro-duodenal junction was healthy and watertight. I examined the excised pylorus. There was neither malignant disease nor ulcer, but there was thickening of all the layers, the muscular layer being enormously hypertrophied.

The method of giving opium in these cases is not unimportant It should be given in doses of from 3 to 10 minims of the tincture in two teaspoonfuls of water twenty minutes before taking food; the dose varying according to the number of meals, and the amount of dilatation. It may be necessary at first to give only liquid food. Opium acts here, as in renal colic and asthma, as an anti-spasmodic for unstriped muscle, and, if given sufficient time to act before food is taken, prevents the spasm of the pylorus. In the motor-neurotic form, food seems to press the trigger for the discharge of the spasm, whilst opium sets the muscle at half-cock.

In the form associated with gastric ulcer, of which a valuable account is given by Habershon,[2] the pain begins some hours after food, the trigger for the spasm in this case is actuated either by the chemical changes in the gastric contents, or by the movements of the pyloric end of the stomach irritating the ulcer. Here opium should be given some half-hour before the usual time at which pain proclaims the onset of pyloric overaction.

It was from the results obtained with opium in the motor-neurotic form in adults that I was led to use the drug in the congenital form. Heubner, however, had previously strongly advocated the use of opium in conjunction with poultices to the abdomen in the congenital form. My own experience is entirely in accord with his view that operation in the congenital form is unjustifiable.

It is important, however, that the drug should be given at the right time, and, when this is done, it is remarkable how small a dose is effective. One minim of tincture of opium is added to 10 ounces of water, and a teaspoonful, that is $\frac{1}{80}$th minim of the tincture, is given twenty minutes before each feed. The vomiting soon ceases, the pyloric tumour disappears, and the infant rapidly puts on weight. Since the cases recorded[3] previously, I have had one typical case, the treatment and the result being the same. A case has also been recorded by Dr. J. W. Rob,[4] in which this method was successfully adopted in conjunction with lavage.

In conclusion, it need hardly be pointed out that it is unnecessary, in cases in which frequent spasm or overaction of the pylorus is suspected, whether in infant or adult, to wait until there are signs of pyloric hypertrophy before giving opium. Indeed, there are many cases in which the spasmodic condition does not lead to pyloric hypertrophy, but which would be more rapidly relieved by the use of opium.

[1] Boas: *Archiv fur Verdauungs-Krankheiten*, Bd. IV., 1898.

[2] Habershon: *Trans. of the Medical Soc.*, Vol. 26.

[3] Neild: *Lancet*, November 25, 1905.

[4] Rob: *Lancet*, June 23, 1906.

ON THE INDICATIONS FOR THE USE OF OPIUM IN ACUTE DISEASE.

By **H. S. SANDIFER, M.D., B.S. (Lond.), F.R.C.S. (Eng.),**

No man can be long in practice, especially general practice, without observing how medicine and psychology overlap each other, and he may finally come to the conclusion that they are simply branches of the one art of curing disease. Nor is he long without observing the beneficent or maleficent influence of mind states upon the body in illness, and therefore upon the course of disease. The emotions of fear are maleficent: those of cheerfulness, beneficent: also, on the other hand, constant physical pain and disease produce distressful mind states, and wear down a disposition that is naturally cheerful and courageous.

That this must necessarily be the case is at once obvious from the consideration of this simple fact, that, in consciousness, each mind state must work a brain state and vice versâ, and that they are inseparable.

Now by means of drugs operating on the brain, especially opium and the bromides, we are able to modify mind states. We can interfere with assaults on the consciousness from below, as it were, by the administration of such drugs, and so protect it from disagreeable afferent impulses, which would produce maleficent mind states; but I think one is often not sufficiently alive to the logically necessary converse, that by the same means one can deliberately, as one often does unintentionally, interfere with the assaults from above, as it were, upon the poor suffering body below.

Thus it appears to me that drugs, acting on the convolutions, may be said to have a psychic action as well as a physic. And that, in one's daily routine in the administration of drugs, one often uses opium and its derivatives for this particular purpose of altering, or modifying, maleficent mind states; and indeed were it not for the action of this drug on the lower centres and distant organs, as well as on the convolutions, one would still more frequently use it. In acute disease, I believe it to be far superior to bromides.

Thus regarding the convolutions as the organ of mind, one may say that the use of opium in acute disease is for three main purposes :—

(i) To modify the emotional state of the patient.
(ii) To diminish or suppress the appreciation of pain and shock.
(iii) Either alone, or more often in combination with pure hypnotics, to produce sleep..

In acute disease, which threatens the life of the patient, *e.g.*, Pneumonia, the importance of the first indication for the use of opium is often the paramount, for the baneful or beneficent effect of mentality, in its aspect of morale, in the course of this disease will at once be apparent.

To compare extremes, one may contrast the condition, say, on the fourth day, of a northern woman who has passed her days in temperate, self-controlled family life, with the condition, on that day, of a highly imaginative, sensitive, and neurotic Jewess, who has possessed the means to indulge her imagination, emotions, and artistic instincts to their utmost.

Now, *cæteris paribus*, their conditions are very dissimilar ; the former is hopeful, manageable, and sub-cheerful, and bears her pains and distress with patience and fortitude, the latter is full of self-pity, and of indignation at her illness ; she describes, in superlatives, the agonies she is suffering, is discontented and fractious, irritable and restless, and is literally "fretting herself to death." In the former case, one has no anxiety as to the issue, in the latter, one feels, one knows that, unless this wear and tear, inflicted by the emotions, can be stopped, a fatal termination is inevitable. Well, if one cannot encourage the beneficent emotions to the exclusion of the maleficent ; to paræsthetise the organ, by which this temperament is being manifested, is the only chance for the patient. This one can do with frequent small doses of opium, say nepenthe, v. x., every few hours, carefully watching the heart and the respiration, especially the former, and, if necessary, giving digitalis, remembering at the same time that the object is to affect the convolutions only, and not to unduly trespass below them. One is often astonished at the rapid improvement in the pulse, and general condition, thus produced by the inhibition of the fear emotions.

To push so far as to bring out the hypnotic effects of opium is too dangerous a procedure. It is in such a case that the excellent effects of combining small doses of opium or its derivatives with simple hypnotics can be noted. For example, $\frac{1}{6}$th gr. of morphine, hypodermically, with 10–15 grs. trional, given in a cachet an hour previously, or nepenthe, ♏xx.–xxx., with bromide, grs. xx.–xxx. In such a condition as this, and indeed in all conditions of moderate pain and emotional disturbance with necessary insomnia, such, for instance, as obtain after operation, combinations like these are very effective. They obviate the inimical effects on the alimentary tract, the often dangerously insensitising effects on the respiratory tract, not to mention the effects on the cardiac and respiratory centres, of the larger doses of opium which are necessary to produce sleep. While the opium dulls the pain and quiets the emotions, it permits the hypnotic to produce sleep.

I believe that this power of opium to act on the personality, viâ the mind organ, is one of its most useful qualities, and that it is therefore a potent instrument in our hands in the treatment of acute disease. Moreover, this effect of the drug is a necessary and useful adjuvant, when it is given for the purpose of producing some other of its effects. In pulmonary or gastric hæmorrhage, for instance, it is probable that the quietened heart and circulation are brought about as much by the diminished emotional perturbation as by the direct effect of the drug upon the circulatory system. With the precautions hereinafter to be mentioned, the drug may be thus used in all acute general diseases, such as the specific fevers, for both adults and children, when the over-anxious, irritable, or timid disposition of the patient is a distinct hindrance to the favourable issue of the ailment; it may also be used in cases of delirium.

I think the question of the acquiring of the drug habit in acute disease can be ignored; besides few lay persons can tell, when Nepenthe, Pulv. Ipecac. Co., Pil. Saponis Co., Tinct. Camph. Co., Heroin Hydrochlor., or Codeina, is ordered, that opium is being administered.

For this psychic purpose, I prefer Nepenthe for adults, and Nepenthe, or Pulv. Ipecac. Co. for children. Leaving the consideration of this special influence of opium on the personality, viâ the mind organ, its use in some of the common acute diseases may now be considered.

In respiratory diseases it stops cough, and for dry useless cough of all kinds, heroin seems to act better than most other preparations, especially in the dry tracheitis associated with influenza. Its use in pneumonia has already been indicated, and in acute bronchitis we are brought face to face with both its advantages and its dangers.

In the dry stages, its use is beneficent, and a hot bath, an unfashionable jacket poultice, and a full dose of Dover's powder make an excellent commencement.

But, in the later stages, when the secretions are abundant, and must be removed by coughing, its insensitising effect on the bronchial mucous membrane alone permits the accumulation of the secretions, and forms a bar to its uninfluenced use. It is, however, just at this period of the disease, especially in old people with copious secretions, that one most wants the influence of opium on the convolutions to produce calm and sleep in order to repair exhaustion. In this quandary, its use, in conjunction with belladonna, presents itself.

Belladonna has a remarkable influence in drying up the bronchial secretions when given boldly. It is also a stimulant to the respiratory centre, thus producing the reverse effect of opium.

I have, therefore, found it to be productive of excellent results to give belladonna in three-hourly doses during the fifteen hours of the day, and then to get the patient under the restful influence of opium during the nine hours of night. In the meantime the heart should be closely watched, and treated, if necessary, with hypodermic doses of strychnine.

In the acute stages of asthma, before other remedies can be got to bear on the lung condition, morphine and atropine, hypodermically, are invaluable for the relief they give to the sufferer. Hot black coffee, always at hand, is a powerful adjuvant, and neutralises any cardiac depressant effect.

Little need be said here of its use in acute diseases of the circulatory system. Hæmorrhage has been mentioned; and in acute endocarditis and pericarditis it slows the heart, diminishes pain and blood-pressure, and relieves anxiety. This also is its action in so many other acute diseases associated with pain, *e.g.*, otitis media, varieties of synovitis, acute rheumatism, etc.

In acute abdominal diseases it is, indeed, a two-edged sword, and diagnosis must be very perfect before venturing to use it. In cases of colic, where confident of its origin, and in biliary or renal calculi, morphine must be pushed to the relief of pain. But when the colic is intestinal, or the diagnosis is uncertain, and operation is not decided upon, I feel confident that it is one's painful duty to withhold opium, and trust to local hot applications and carminatives, especially chloroform, by the mouth. Of course, when one has made the diagnosis of strangulated gut, intussusception, perforation of appendix or stomach, or the like, or where one has made up his mind, from the urgency of the symptoms, that the abdomen must be opened, then morphine may be given up to the point of relief of pain; but even then it is best given with atropine, and in the minimum effective dose, so that the patient may be saved as much pain as possible from subsequent post-operative flatus aggravated by paresis of the gut. One cannot too forcibly insist that opium is capable of entirely masking every symptom of acute intestinal lesion. I have seen a man, at 9 p.m., who, a few minutes before, had a perforated appendix, rolling in his bed with agony and sweat, vomiting, and screaming for his gun to blow out his brains, and who, after ½ gr. morphine, at 11 p.m., was placidly lying in his bed, waiting for operation, with temperature 99 and pulse 72, and a flat abdomen almost free even from tenderness.

In the treatment of acute disease in children, it is doubtful whether we use opium often enough. It is true that children are especially susceptible to the drug, but it is generally only a question of dose. I use it almost as freely, and for the same purposes, as in the cases of adults. It should, however, be given with the following precautions:—Give very small doses frequently, and state, in both the written and the verbal directions, that the child must never be wakened to take its medicine. An overdose is thus impossible. It is of great value to timid, highly strung children, especially in the form of small doses of Dover's powder in feverish conditions.

In conclusion, I should like to emphasise two other points, (1) to learn the state of the kidneys of your patient; (2) be sure that he has no idiosyncrasy before using the drug. I think a large proportion of people have an idiosyncrasy against opium. I once injected, late at night, $\frac{1}{6}$ gr. of morphine, and $\frac{1}{150}$ gr. of atropine, into a lady of 24 years, who was suffering acute pain. I spent the rest of the night keeping her going with brandy and strychnine. She nearly died. Subsequently, I explained the importance of the certain knowledge of her idiosyncrasy, and advised an experiment. Accordingly, one hour after her breakfast, I began with $\frac{1}{12}$ gr. of Morph. Hydroch. by the mouth. In less than five minutes, a deathly pallor and nausea overcame her, she broke out into a cold, clammy sweat, and her pulse dropped from normal to 40 feeble beats per minute. $\frac{1}{60}$ gr. of strychnine and brandy brought her round in two hours. I congratulated myself that I had not given, hypodermically, $\frac{1}{4}$ gr. instead of $\frac{1}{6}$ gr. of morphine as her initial dose.

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OPIUM IN ACUTE DISEASES.

By ARTHUR BOUSFIELD, B.A. (Cantab.), M.D., B.Sc. (Lond.).

In considering the indications for the use of opium in acute diseases, it is necessary to take into account, on the one hand, its specific actions, the more important of which are seen in its power of checking the peristalsis of the intestinal tract, and of lessening the irritability of the respiratory centre; and, on the other hand, what may be described as its general action—that is, its ability to relieve pain, in whatever part of the body it may occur, and from whatever cause it may arise.

It is in connection with this latter function that there is much difference of opinion, and somewhat divergent views are held as to when opium should be used to relieve the various painful conditions met with in the different forms of acute disease. It is, therefore, principally on this point that I venture to offer a few remarks.

In all kinds of acute disease, when a decision has been arrived at as to the nature of the illness, and as to the main lines upon which it shall be treated, the very important question arises: in what manner can the patient be most quickly relieved from his immediate sufferings, so that he may be made as comfortable as circumstances permit, and be enabled to obtain a fair amount of sleep?

In order to determine whether this object shall be attained by the administration of opium it will be advisable to consider—

(i) The nature of the suffering, whether the pain is really severe, or whether, with a certain amount of pain, the condition may not, in the main, be due to excitement, anxiety, or restlessness.

(ii) Whether the pain can be thoroughly relieved by other means.

(iii) If it is determined that the pain cannot be satisfactorily relieved without opium, whether there are any contra-indications which would nevertheless make it necessary that opium should be withheld, or given in a greatly reduced quantity.

(iv) If opium is to be given, in what form shall it be administered, and in what dose, and what are the indications for giving, or withholding, subsequent doses?

(i) In some cases the patient may exhibit signs, which will at once indicate that the pain is severe. The face may be drawn and pinched, with perhaps beads of perspiration on the forehead. The pulse may be small, rapid, and thready. He may be lying on his back with his legs drawn up, or with one arm pressed tightly to his side; he may speak in a faint voice, with perhaps a gasp between every few words. And this first impression that the pain is really severe may be confirmed by the result of the systematic examination of the various signs, which are presented by the particular disease from which he is suffering.

On the other hand, the patient may present a very different picture.

The face may be hot and flushed, the pulse full and bounding. He may be restlessly turning and twisting in bed, tossing the clothes about, talking excitedly; and may, perhaps, with some volubility, describe his sufferings as almost unbearable; while the physical examination may not reveal signs of any of the morbid conditions usually associated with, or giving rise to great pain.

In the former case only, opium in some form would be of any avail, whilst, in the latter, some such remedy as a chloral and bromide draught, or trional gr. xx., would be more appropriate.

In intermediate cases, where, with a great deal of restlessness, there is a moderate amount of pain. Liq. Morphinæ hydrochlor, ♏x. xv. may, with advantage, be added to such a draught.

(ii) The pain may be fairly severe, but it may be possible to relieve it by means of other drugs, or certain remedial measures, without recourse to opium; and, in such cases, the preference should be given to these other remedies, and resort be only made to opium, should they fail.

For instance, salicylate of soda will, in almost all cases, relieve the joint pains in acute rheumatism, while Vin. Colchici will do the same in acute gout.

Severe headache and pains of a neuralgic character will be best treated by phenacetin, or some drug of a like nature.

The sharp muscular pains in back and limbs, so often met with in influenza, if not relieved by quinine, will often yield to a full dose of aspirin.

Three or four leeches may obviate the necessity of giving opium to a patient suffering from an acute attack of either pleurisy or pericarditis.

A large mustard and linseed poultice will sometimes, with great rapidity, relieve severe pain in whatever region of the body it may be felt.

In such cases as the above, these alternative remedies should have a fair trial; usually, they will do very much more towards removing the cause of the pain than will opium; and, with most of them, there will not be the fear that, as soon as the effect of the drug has worn off, the pain may return.

(iii) There are certain contra-indications, which must be carefully considered, when giving opium in acute diseases; some precluding its use altogether, whilst others will suggest that only a very small quantity may be given with safety.

If the lungs are embarrassed with a considerable amount of fluid, of either an inflammatory nature, arising from diffuse bronchitis, or broncho-pneumonia, or serous, as met with in the œdematous bases so often complicating any acute disease in elderly people, or in those suffering from weakness of the heart's action from whatever cause, opium should not be given.

Any ease resulting from its administration, being too dearly purchased at the risk, that, after the rest from excessive respiratory effort so obtained, the lungs may not be able to free themselves from the extra amount of fluid thus allowed to accumulate.

The sensitiveness of young children to even very small doses of opium is so constant, that everyone is agreed that it should be avoided, as a rule, in the treatment of their acute diseases, except in certain cases; for instance, in Cholera infantum, where the specific action on the intestines may be invaluable.

In older children, opium should be given in very moderate doses, and its action carefully watched.

The same may be said with respect to those well advanced in years; and it is especially necessary to bear this in mind, because it is not uncommon for an elderly patient, when suffering from a painful complaint, to assure his doctor that, in a previous attack of the same nature, he has had a hypodermic of morphia, perhaps mentioning that he had been told that a sixth or a quarter of a grain had been given him; and, at the same time, saying that he feels sure that a similar injection is the only thing which will do him any good. He may be quite accurate in his statements, even to the dose given, but the ten or fifteen years which may have elapsed since the previous attack may, perhaps, have so altered his general condition that, instead of the sixth of a grain demanded, it may be advisable to give a twentieth, and this minute dose will often be perfectly successful in satisfying the patient and relieving the condition.

With regard to patients with high arterial tension, and other signs of chronic renal disease, the fact that morphine is excreted almost entirely by the bowel, and scarcely at all by the kidneys, and that it can be given in uræmia with safety, might be taken as showing that the condition would not necessarily contra-indicate the use of opium.

This may be the case when the disease is very strictly localised, as, for instance, in some limited acute inflammation or spasmodic contraction of a muscular tube; but when the acute disease is of a general or infective character, there is certainly, in cases of chronic renal, a special danger that respiratory complications, such as diffuse bronchitis or œdema of the lungs, may supervene; and, for this reason, opium should be avoided, or used with great caution.

Digestive disturbance, headache, and a very constipated condition of the bowels are, in lesser degrees, contra-indications, as there is a tendency for opium to aggravate all these conditions.

When, in any acute abdominal illness, the question of operation may have to be raised, care must be taken to give opium, in such moderate doses, that, while the immediate pain is relieved, the clinical picture is not obscured, and the chances of success, should an operation have to be performed, are in no way lessened.

(iv) When opium is given in acute disease, with the alleviation of pain as the primary object, it is an advantage that the effect should be obtained quickly, and with as little interference with the digestive apparatus as possible. This can be best achieved by the administration of morphia hypodermically, and for an adult, between 20 and 60 years of age, gr. $\frac{1}{4}$ of Morphinæ sulph., with gr. $\frac{1}{100}$ of Atropinæ sulph., should usually be given. Various considerations, mentioned above, will make it advisable to reduce the dose to gr. $\frac{1}{6}$ or gr. $\frac{1}{8}$, or, in special cases, to considerably less than this.

If, at the end of an hour, the pain has not been relieved, it is permissible to repeat the hypodermic, giving, as a rule, the same dose. If some relief has been obtained, it will be best either to wait another hour, or to order a draught containing a quantity of Liq. Morph. Hydrochlor., corresponding with the original hypodermic, half to be taken at once, and the rest, if required, in two hours' time.

In cases in which, besides for the relief of pain, it is desired to make use of the specific action of opium on the bowel, or respiratory apparatus, other preparations may be more suitable.

Thus Pil. Saponis Co., or Liq. Opii Sed., may be given for pain of moderate severity, when there is enteritis, or peritonitis, and a continued action on the bowel is desired.

In the early stages of bronchitis, and in laryngitis, so often associated with influenza, instead of the 5 or 10 grain dose of Pulv. Ipecac. Co., heroin may be used for relieving the constant irritating cough, and so inducing sleep.

℞.	Heroin Hydrochlor. - -	gr. $\frac{1}{8}$.
	Acidi Hydrocyan. dil. - -	♏viij.
	Aq. Chloroformi - -	ad ℥ij.

A tablespoonful to be taken every hour until relieved, will usually give good results, and will not cause any headache, or nausea, the following morning, such as not infrequently occurs after taking Pulv. Ipecac. Co.

In prescribing opium in any form, in acute disease, it should be felt that the physician has convinced himself that every dose which is given is really a necessity. In this way only can it be assured that this powerful remedy will be used with safety and success.

A REVIEW OF RECENT NEUROLOGICAL SURGERY.

By DONALD ARMOUR, M.B., F.R.C.S.,

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CUSHING (*Amer. Jour. of Med. Sciences*, October 1905) has an interesting paper concerning surgical intervention for the intracranial hæmorrhages of the new-born. Various terms are used to indicate those infants who give evidence of serious cerebral defects that have been acquired during infancy, these terms being descriptive merely of a common residual symptom —the unilateral or bilateral spastic palsy.

It is generally stated by the many writers on the subject, that in a cerebral vascular lesion, hæmorrhage is, in the large majority of cases, the causal factor of the malady which is of primary importance. This applies to the cases of pre-natal, as well as those of post-natal origin, but more particularly to those incident to parturition, the so-called birth palsies. It is with this last group that Cushing's paper primarily deals.

He points out that the intracranial hæmorrhage is usually of venous origin, and follows the rupture of some of the delicate and poorly supported venous radicles of the cerebral cortex. Such an injury may be the direct result of undue traumatism during labour, or may occur when too great a strain has been put upon the vessels by the profound venous stasis of postpartum asphyxiation. Cushing had been struck, during an investigation into the circulation of the cerebro-spinal fluid, by the considerable percentage of dead infants who showed an intracranial hæmorrhage. That one of these cortical hæmorrhages had been the cause of death could not always be proved, though, in some of them, the effusion of blood was so great that little doubt was felt that it must have played an important part in the asphyxiation which the clinical histories recorded. As Cushing points out, when one considers the immense amount of traumatism to which the infant's head must necessarily be subjected, particularly should the child be large, or the first-born of an elderly mother, it is little to be wondered at that lesions of this kind occur. The infant's brain itself is doubtless fitted by nature to withstand, without injury or shock, a degree of moulding and manipulation which could not be borne by a fully developed and medullated adult brain. Cushing thinks, from his own studies, that the vessels most likely to rupture are those which ascend over the cortex, and enter the longitudinal sinus from the mid-cerebral region. These veins which lie in the sub-arachnoid space, and for the most part are protected from rupture by following the sulci as they ascend towards the middle line, leave this protection when near the vertex, and, apparently with no support whatever, cross the subdural space to enter the dura at the outer edge of the sinus. In infants, these unsupported vessels are the only connection between the leptomeninges of the hemispheres and the dural sheath underlying the sinus. Although the cranial moulding may take place to a considerable extent, without putting any great strain on these lateral cerebral veins, any undue overlapping could readily tear one or more of them away from the sinus. Cushing thinks that this is the more common form of injury, and that it possibly accounts for the fact that the primary effusion, and therefore the thicker portion of the clot, is commonly median, and in consequence implicates primarily and most seriously the centres for the lower extremities. He thinks that the extravasation is usually limited to one side of the falx, though indeed a bilateral lesion is common enough, as the many patients with diplegia would indicate, and is of opinion that the condition should not be difficult to diagnose. Should there be any serious doubt as to the actual nature of the lesion, it might be decided by lumbar puncture, when free corpuscles will be found in the fluid of the lumbar meninges. This would serve to differentiate between hæmorrhagic and infectious cases, where diagnostic difficulty is present.

Cushing points out that the history of the labour is all-important, and also the degree of post-partum asphyxiation. Among the objective signs he places first the condition of the fontanelles. The membrane is bulging, and in some instances may be so tight as to show no pulsation. Convulsions are frequent, and, particularly if they do not appear until several days after birth, they may be more or less unilateral in character. There may also be an undue reflex activity, the child starting or twitching in a convulsive way in response to any sensory stimulus. Ocular palsies, or differences in the size of the pupils, are not uncommon; and, in severe cases, there may be symptoms referable to the medulla, such as alterations from the normal in the cardiac and respiratory rhythms. Evidences of paralysis are rare.

Putting aside the fully-developed and terminal aspects of the disease, there is one very serious sequel, which frequently, according to Gowers in two-thirds of the cases, accompanies the mildest grades of the disease, even when intelligence may in nowise suffer. This is epilepsy, and the convulsions, often Jacksonian in character, are akin in all respects to those which follow the cortical changes resulting from cranial injuries with hæmorrhage in adult life.

Cushing reports four cases operated upon by him, two of which were successful. An osteoplastic flap was raised, which included the entire parietal bone. The tense dark plum-coloured dura was then brought into view. On opening this membrane a clot was disclosed. It was lifted off in large pieces from the exposed area of the cortex with a blunt instrument, and a large amount of clotted blood, which extended over the frontal and occipital regions, was irrigated out from under the dura with warm saline solution. The wound was closed without drainage.

In one of his cases which recovered the operation was done on both sides.

He draws the following conclusions from his experience in these cases:—One is, that a new-born child, with a proper regard for hæmostasis and careful avoidance of exposure, will stand a cranial operation well, though this is contrary to the statements in our text-books. An argument advanced against the possibility of carrying out these operations on the new-born is that the blood of these infants has such a slow coagulation time that a fatal hæmorrhage from the incision would result. Cushing says that this is no more true for cranial operations in the new-born than for those in other parts of the body, and his experience leads him to think that coagulation is as rapid as in the adult. It is worthy of remark in this connection that the effused blood, in all of his cases, was found clotted. In three of them, those with primary cortical hæmorrhages, the effusion was confined to one side.

He concludes that if it can be demonstrated that a craniotomy on the new-born child, when conducted with due precaution and delicacy of manipulation, is comparatively free from danger, the immediate risk of death, and the sorry later consequences of meningeal birth hæmorrhages, may be avoided, in many cases, by surgical interference.

In a discussion, before the Boston Society of Psychiatry and Neurology, on the "Indications for Operative Treatment in serious Head Injuries," Sachs (*Boston Med. and Surg. Journal*) lays stress upon the following points:—

1. If there is extensive injury to the skull, particularly in the parieto-temporal region, whether fracture or fissure; if there is evidence of splintering of the inner table, or of the presence of a foreign body, or of persistent intracranial hæmorrhage, operative interference is warranted at the earliest possible moment.

2. In comminuted fracture of the skull, the surgeon must decide whether or no the danger of infection is increased by surgical procedures.

3. In all cases, but especially in those in which external injury cannot be taken to be a determining factor, the question of surgical interference must be decided on purely neurological lines.

4. He thinks it useless to continue the differentiation between concussion, contusion, and compression, and considers that it is more important to decide whether the brain has or has not been tangibly injured, and if injured, whether the site of the injury is accessible or not. If inaccessible, simple trephining may be resorted to, provided there are symptoms of increasing intracranial pressure, which cannot be relieved by lumbar puncture, or other similar methods.

5. Even if the injury is in an accessible region, it is best to adopt a conservative attitude, and to determine whether we may trust to surgical skill rather than to the reparative powers of nature.

6. In determining the gravity of brain injury, disturbances of cardiac and respiratory action, and of vesical and rectal control, and the condition of consciousness, are the most important symptoms. They are the manifestations of increasing intracranial pressure, and of other serious injury. Recovery from coma is encouraging; deepening coma is of grave signifi-

cance. The behaviour of the pupillary reflexes is of no special value in deciding the question of operative interference.

7. If the symptoms point to a distinct focal lesion, surgical measures must be adopted, provided that the lesion is accessible.

8. If the external injury points to one side, and the symptoms to the other, consider both. Take the side of external injury first, but try to reach the other as well.

Prince, continuing the discussion, says that it is necessary to take into consideration four kinds of evidence in deciding whether we have to deal with a hæmorrhage, or laceration, or contusion of the brain, or a combination of the three, and the extensiveness of the lesion. They are: —(1) The nature of the blow; (2) The general symptoms, such as coma, temperature, pulse, stertor, etc.; (3) The neurological indications; (4) Surgical evidence of injury to the skull.

Bullard thinks that, in speaking of the indications for operation in head injuries, only certain general lines of treatment can be laid down. There must be exceptions to the general rules in certain cases, and to some of the rules in many cases. In compound fracture of the outer surface of the cranium, he thinks that operation in adults is always advisable, assuming that there are no serious contra-indications from the general condition of the patient. Probably, even in long linear fractures, where there is no displacement or depression of bones, it is wiser to operate. In simple fracture of the external surface of the skull in adults, it is safer to operate. In children, it is sometimes permissible not to operate where no symptoms exist. All depressed fractures should be operated upon. Operate in all cases in which symptoms of middle meningeal hæmorrhage exist. He would, therefore, as a rule, operate in any case in which unconsciousness comes on after an interval of consciousness, following injury to the head. Operate in adults whenever the unconsciousness, after severe head injury, lasts more than twelve hours, and when it seems clear that the unconsciousness is due to the injury, and not to alcohol, or other causes, or complications. He admits, however, that this rule is not universal. As a rule, it is wise to operate when persistent unilateral convulsions follow injury to the head in an adult, provided that such convulsions have never occurred previous to the injury, and that no other cause for them exists. When cerebral or meningeal paralysis occurs immediately after a severe injury to the head, the question of operation often arises. Hemiplegia, or monoplegia, in such circumstances, does not absolutely indicate operation. They may be due to hæmorrhage, or other injuries in parts of the brain which are out of reach. Inequality of the pupils, occurring immediately after an injury, is an indication in favour of operation. Immediate and persistent rise of temperature, not otherwise accounted for, occurring after severe head injury, suggests contusion or laceration of the brain. A rise of temperature, even 24 to 72 hours after injury, especially if the patient is unconscious, suggests secondary encephalitis, or inflammation of the brain. A slow pulse suggests compression, and, therefore, with other symptoms, is an indication for operation. Rapidity of the pulse in itself does not contra-indicate operation. Weakness of the pulse may be a contra-indication.

Walton was of the opinion that the following symptoms in themselves were not sufficient to justify operation:—(1) Continued unconsciousness, not deepening; (2) paralysis appearing immediately, and not increasing; (3) inequality of pupils; (4) delirium and restlessness; (5) the presence of fever. Among the conditions indicating operation he included:—(1) Deepening initial unconsciousness, or unconsciousness coming on after an interval, and deepening into coma; if no other localising sign accompanies, the seat of injury should be chosen for operation. (2) The spread or increase of initial paralysis after an interval. This points to hæmorrhage. In the presence of this condition, the possibility of bruising and œdema alone being present does not suffice to contra-indicate operation. (3) Jacksonian attacks. (4) Dilated pupil on one side, accompanied by paralysis or spasm upon the other. (Middle meningeal hæmorrhage.) (5) Punctured or definitely depressed skull. (6) The question whether simple fissures and moderate depressions of the skull require operation demands surgical, rather than neurological judgment. The benefit of the doubt should be given to operation, in view of the many cases, in which exploration of such injuries reveals widespread and serious conditions likely to prove a future menace.

REVIEW OF THE DISEASES OF THE RESPIRATORY TRACT.

By J. J. PERKINS, M.B., F.R.C.P.,

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"The Role of Saline Solution in the Treatment of Pneumonia."[1]

THE use of injections of saline solution for pneumonia was first introduced, the author says, by F. P. Henry in 1889, the idea being derived from its value in surgical practice. Since then, this mode of treatment has been occasionally adopted, either alone or in conjunction with bleeding and oxygen inhalations, but, as a rule, has been restricted to desperate conditions. The grounds on which the author would see saline infusion used are, however, quite different from those on which it was first suggested. Instead of waiting until the patient is saturated with toxins and moribund, he would have recourse to it in anticipation of those symptoms which, following Sajous, he believes can be directly prevented by its use. The argument on which this view is based is as follows:—

Recovery from pneumonia, or any other infectious disease, depends on the formation of protective substances, the "immunising anti-bodies" of Behring, which appear in the blood wherever the exact seat of their origin may be, and determine the contest with the invading organisms. For their production, good nutrition, and vigour of the tissues are necessary. Now the health of the tissues is dependent on the presence of alkaline salts in the blood, and is impaired when they are deficient. The author quotes Lazarus Barlow to show that the alkalinity of the blood is diminished during fever, and that, with diminution in alkalinity, goes hand in hand an increased susceptibility to infection. Indeed he says it has been proved experimentally that saline solution can maintain the blood's immunising power.

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[1] Madison Taylor: *Med. Record*, January 13, 1906.

Some 200 grains of sodium chloride are voided daily in the urine, and this amount cannot in fever, owing to the restricted diet, be replaced. The diminution of the chlorides in the urine in pneumonia, the author looks upon as an attempt of the organism to hoard up the necessary salt, but even so it is consumed and inadequately replaced. A stage is finally reached in which the tissues are so impaired that they cease to secrete the necessary protective substances, and the pathogenic organisms have full sway.

M. Taylor would therefore, without waiting till things become desperate, give saline injections in all fevers from the outset, in order to ensure the nutrition of the tissues, and keep up an adequate formation of the protective substances, thus preventing the organism from becoming loaded with toxins.

As far back as 1898, Lemaire, some of whose results are quoted, was using this mode of treatment early in the course of the disease, as soon as the usual remedies failed to produce improvement. His theory was that of washing out the blood, but analysis shows that, under saline treatment, the urine becomes not more but less toxic, because, according to our author, the toxins are not washed out but destroyed in the blood. It is claimed that by saline injection the blood-pressure is raised and diuresis increased ; general improvement follows, though the temperature may rise for a few hours after injection.

At the Roosevelt Hospital in New York, normal saline solution, ℥viij. 4tis horis, is injected into the rectum ; the results are reported as good.

Recently, Todd has introduced the method of giving it as a lemonade, ℥vj. - viij. secundis horis for adults, each ounce containing gr. x sodium chloride, gr. v. potassium bicarbonate, with ʒi. lemon juice.

Madison Taylor's paper was read at a Symposium on Pneumonia of the Medical Association of the Greater City of New York, the increasing spread of pneumonia in America making it a serious question. It is interesting to note the measures advocated at this Symposium. Anders considered that the sputa were the chief source of infection, and urged their thorough disinfection and destruction.

Loomis had collected the details of treatment in four of the large New York Hospitals, and had found, on the whole, an unexpected uniformity. Local applications are used only for the relief of intense pain. No effort is made to reduce the temperature, even at 104°, unless the fever is accompanied by nervous symptoms or delirium : when some form of cold-water application, or alcohol sponging, is employed. As cardiac stimulants, alcohol, strychnine, and digitalis are chiefly used at all four, the first being most relied on, and the last-named being in use to any extent at the Roosevelt Hospital only. Oxygen is given when there is cyanosis and dyspnœa, but apparently with little benefit.

Saline injections, as stated above, are mentioned as in use at the Roosevelt Hospital ; the results are reported as good.

Loomis strongly advocated morphine when there is severe pain, and held that two points in the usual treatment are most open to criticism, the injudicious and unwarranted use of alcohol, and, possibly more important still, the promiscuous drugging which is still too prevalent.

In this last point he was supported by Hare, who deprecated the routine use of drugs from the start, preferring to reserve them for special indications. Atropine he finds valuable ; digitalis is inefficacious, partly owing to the fever, and partly to the enfeeblement of the myocardium.

Northrup and Page insist on the importance of the freest ventilation in the treatment of pneumonia, and condemn the stifling conditions, which are too often found in the sick rooms even of the wealthy.

The former (*Boston Med. and Surg. Journ.*, February 22, 1906), under the title of "The Cold Fresh Air Treatment of Pneumonia," reports the recovery of a case which has evidently attracted great attention in the States, and its lesson is a salutary one. The case was a severe one of wandering pneumonia, involving ultimately the whole of the left lung, with high fever (104°-105°) and violent delirium. Though the month was March, the windows were thrown wide open, and the head of the bed placed between the two open sashes of a bay window ; the temperature of the room was about 40° F.

Page (*Med. Record*, December 23, 1905) has some very interesting remarks on "therapeutic fasting" in the fevers. In his practice, he never allows the ingestion of a morsel of any sort of food, except water, until the patient is practically convalescent. The grounds for this abstinence he finds, of course, in the impairment of the digestive powers.

Fasts of five, eight, and ten days have been so common in his practice that they have long ceased to be novel. Water, moderately hot, he allows in abundance to preserve the integrity of the tissues. While one may not go so far as this in withholding food, it is well to have this possibility of the futility, with its attendant discomfort and danger, of much of the feeding which goes on in acute illness, brought home to one. Everyone who has had much post-mortem experience, and has noted the overloaded stomach so commonly seen, still distended with the milk poured down by assiduous attendants, will have no doubt on this point.

Anders (*Med. Record*, July 7, 1906) also bears testimony to the benefit of cold fresh air in the treatment of pneumonia, with certain exceptions, however, *e.g.*, when the pneumonia is a complication of advanced Bright's disease. His personal experience of the method is confined to five cases, too few, as he allows, for trustworthy inference, but the result in them was so favourable as to encourage its more general adoption. He has not noticed any tendency to "cold," or the development of bronchitis with its use, although he mentions that, in one case, the temperature was so low that the nurses had to wear wraps. He enumerates the beneficial results as "better general condition, improved appetite and digestion, refreshing sleep, diminished fever, breathing and pulse rate. The nervous system partakes markedly in the general favourable effects." He attributes these results partly to the purity of the air, partly to the abstraction of heat, and partly to the tonic effect of cold. The response to the action of cold, it must be remembered, is not always the same in different individuals, and the patient will need careful watching in this respect.

Morris (*Med. Record*, July 1905) once more calls attention to the well-known **simulation of appendicitis**, or other acute abdominal lesion, **by pneumonia**, and the difficulty in diagnosis that may thus be caused, abdominal pain of sudden onset with vomiting occurring in both. Where the physical signs of the pneumonia are delayed, as they may be for some days, the difficulty, of course, is greatly increased, but, even then, the absence of really acute tenderness on pressure in the iliac fossa, on the one hand, and the high temperature and high-pulse rate on the other, are usually sufficient for diagnosis.

Morris lays great stress on the accurate correspondence between the temperature and pulse rate in pneumonia, whereas, in abdominal lesions, a high-pulse rate may co-exist with comparatively slight fever. In one case which he reports, even this criterion failed him : great rigidity of the abdominal wall was present with pain referred to the right iliac fossa and vomiting ; temperature 100° F., pulse 130. A specially careful examination of the lungs revealed nothing abnormal. The abdomen was opened only to find the appendix healthy, and, next day, there were signs of a right lobar pneumonia.

"**One-day pneumonia,**" under this name, Leube (1877) described a rare variety of pneumonia, in which a perfectly typical crisis with free sweating occurred as early as 24–48 hours from the onset.

Nordmann (*Gaz. des Hop.*, June 19, 1900) describes an example, while Bechtold (*Münch. Med. Woch.*) has succeeded in collecting 10 instances (among 1057 cases of pneumonia) from the *Wurzburg Clinique.*

Among other authors, who have recognised the type, are Weill, Steiner (23 cases), and Juergensen (9 cases).

Bechtold, analysing his ten cases, found that an initial rigor was present in all, and that all ended by crisis.

The physical signs, however, were very variable, crepitant rales being apparently the commonest. Bronchophony was present in four, bronchial breathing in two only ; in some, there was well-marked dulness, in others, good resonance was present.

It is important to note that the sputa are not characteristic, rusty expectoration having been observed in one case only.

Bechtold asks whether these are cases of abortive pneumonia, which cease to develop at a certain stage owing to increased resistance on the part of the patient, or whether a pneumonia can pass through all its usual stages in twenty-four hours. No answer can be given to this question as the prognosis is invariably favourable.

Nordmann, however, draws a sharp distinction between this group and abortive pneumonia. According to him, in the latter, the disease, as a whole, comes to an end: in one-day pneumonia, the fall of the temperature is the only change, the physical signs and the general symptoms continuing.

This leads him to suggest that afebrile pneumonia is really one-day pneumonia, which has come under observation after the temperature has dropped.

Several cases have been reported testifying to the value of inhalations of **nitrite of amyl**, first introduced by Francis Hare, (*Australas. Med Gaz.*, 1904), in checking *hæmoptysis*.

In Rouget's case (*Bull. et Mem. de la Soc. Med. des Hop.*, April, 1905) free hæmorrhage had been continuing for four days, in spite of treatment by morphine, ergotine, calcium chloride, etc.

On the morning of the 5th day, ♏x. of nitrite of amyl were inhaled; the hæmorrhage was less abundant throughout the day, and, in the evening, the inhalation was repeated. The bleeding ceased, and from that time forward, only old blood was expectorated. In this case, the value of the drug was proved on five separate occasions; nine successful cases are reported in all.

Lemoine reports two cases, in one of which, hæmoptysis, which had resisted all treatment for three days, was stopped by two inhalations. A fresh hæmorrhage eight days later yielded to three inhalations.

Soulier, Pic, and Petitjean (*Lyon Med.*, 1905, No. 49) report a similar series of four cases of its successful use in large, recurrent hæmoptyses. The dose used varied from three to six or nine drops according to the severity.

They investigated its action experimentally on curarised dogs, the lung being exposed, and the drug injected into the femoral vein.

An intense anæmia of the lung was caused, lasting ten minutes, marked by a change of colour from the natural rose to slate coloured, or white.

While the general blood pressure fell, they found that the pressure in the main pulmonary artery was raised, and from this they infer that nitrite of amyl, while acting on the general circulation as a vaso-dilator, has on the pulmonary system the reverse effect, *i.e.*, acts as a vaso-constrictor.

No ill results have been noticed to follow its use, and it is claimed that it is not only safe, but rapid in its action.

Lawrason Brown (*Albany Med. Annals*, April 1906), however, makes the criticism that it is very evanescent, and, for this reason, prefers nitro-glycerin (long ago advocated by Flick), which he injects hypodermically, or sodium nitrite, which, while slightly less powerful, is a little more prolonged in its action than nitro-glycerin. It too can be given hypodermically (dose one grain). Nitrite of amyl, in addition to its rapid action, has the advantage that it can be put into the hands of the patient himself.

Lawrason Brown advises the administration of sodium nitrite with morphine just after the nitrite of amyl. The blood pressure, he says, may be reduced to too low a level, and needs to be carefully watched.

Sir W. Macewen (*West London Med. Journ.*, July 1906), in his Cavendish lecture, gave a remarkable instance of **drainage of a tubercular abscess cavity** in phthisis, when the condition must have seemed almost hopeless.

The patient was suffering from *orthopnœa*. The left side of the chest bulged, was motionless and quite dull; the heart could be seen beating to the right of the sternum; the breath sounds, vocal fremitus, and resonance were all completely lost.

Expectoration had been free till eleven months before, but had then ceased, and, since that time, the distress and shortness of breath had come on.

The diagnosis was that of a large abscess cavity in the left lung, or pyopneumothorax, which had ceased to empty itself on account of occlusion of the bronchus.

A couple of inches were resected from the eighth and ninth ribs in the posterior axillary line, and the pleura penetrated by dressing forceps. 160 ounces of pus teeming with tubercle bacilli, and containing sloughs of lung and caseated débris, were evacuated, the whole lung having been destroyed. The operation gave great relief, but the cavity, after four weeks, showing no signs of closing, and still discharging freely, an extensive thoracoplasty was done, portions of rib, from the third to the ninth inclusive, being removed from the angle to the costal cartilage. Gradually all the structures presenting on the left side of the mediastinum became covered with and fused together by granulation tissue. Finally, the skin was laid over the pericardium, diaphragm, and stump of lung, and, soon becoming adherent, filled up the cavity, a small sinus only persisting at the upper part.

It is now 11 years since the operation, and during all that time the man has been able to work regularly as a light porter, and support his family.

On June 3, 1900, the patient was examined: the right lung was found to be perfectly healthy and the heart sounds were good. No breath sounds, except such as were transmitted, were heard on the left side. For the purpose of the examination, the man mounted 80 odd steps at the hospital, without a pause. He can walk 10 miles without fatigue, but does so at what he calls "an ordinary easy rate."

Hare ("Food Factor in Asthma," *N. Y. Med. Journ.*, September, 1905) applies his theory of "hyperpyræmia," to the causation and treatment of *asthma*.

Hyperpyræmia is the name he has devised for a condition, in which the carbonaceous intake (*πυρειον*, fuel) is greater than the demand of the tissues, and in which, therefore, the blood becomes overloaded with unoxidised carbonaceous material.

The accumulation gradually increases until a climax is reached, and the excess is used up in the severe muscular work of the asthmatic paroxysm.

In this way Hare explains the relief which an attack may bring to an asthmatic, so that, after the paroxysm, he may freely expose himself to the ordinary causes of his attacks, without exciting one, as if, in Hyde Salter's words, the attack were a sort of "clearing shower."

The valuable effects of physical exercise in warding off the attacks are due to the same cause.

Hyde Salter mentions that some of his patients gave up all treatment, except severe muscular exercise, and Hare finds exercise, after the last meal of the day, very beneficial.

Herein also lies the explanation of the "Sunday asthma" of the working man.

Hyperpyræmia, naturally, is at its height during sleep, when combustion is at its lowest; hence the constancy with which asthma appears in the early morning hours, and in night-workers, according to Hare, its predilection for the day.

The relation of asthma to meals, he finds to be due to the actual presence of increased food material in the blood, and not to gastric irritation. In accordance with this, as Salter noticed, we find rapidly absorbed substances capable of exciting an attack almost immediately, while, with the more slowly absorbed, the attack may not come on for a couple of hours.

As to treatment, his theory would lead Hare to restrict the food supply, and the starches, sugars, and fats, rather than proteid material. He quotes some instances of the successful treatment of asthma on these lines. He is careful, however, to make the reservation that hyperpyræmia, though present in most cases, is only one factor. The special vasomotor irritability, which is the characteristic of the asthmatic, must also be taken into account, and this may predominate.

Consequently cases cannot always be successfully treated by dieting only, and attention, he allows, may even need to be directed to the vasomotor condition first.

S. T. Darling (*Journ. Amer. Med. Assoc.*, April 28, 1906) reports a case, in one of the negro labourers working on the Panama Canal, of **general protozoon infection**, producing, among other lesions, pseudo tubercles in the lungs.

The patient, who had been ill nearly three months, was admitted to hospital with fever and delirium, and died the day after.

At the autopsy, the lungs were found to be studded with pale grey nodules resembling miliary tubercles: the spleen, which was much enlarged, contained a number of somewhat similar nodules, except that they were yellow. The mesenteric glands were enlarged and pale, and the peribronchial lymph nodes contained a few recently caseated tubercles.

The post-mortem diagnosis was acute miliary tuberculosis of the pulmonary type, but a fuller examination revealed a very different condition of affairs, the peribronchial lymph nodes alone being tubercular, true tubercles and tubercle bacilli being universally absent elsewhere.

On section and microscopic examination, the pseudotubercles in the lung proved to be areas of catarrhal pneumonia, the desquamated cells lying in the alveoli being filled with myriads of parasites.

Parasites were present also in the alveolar epithelium where it still lined the walls, but were only rarely seen lying free, showing, not only in the lungs but throughout the body, a predilection to invade epithelial and endothelial cells, but never the red-blood corpuscles. The lung itself played a passive part, there being no leucocytic infiltration of the pneumonic areas. The nodules in the spleen were due to the same agent, which, in the liver, had produced areas of necrosis by invasion of the hepatic cells, but more especially of the endothelium lining the capillaries. The parasite was present in the mesenteric glands and in the bone marrow. Under a high power, it appeared as an oviform, or round body enclosed by a clear non-staining refractile rim, and, while resembling some of the other protozoa already described, presented certain peculiarities. Since writing this article, the author has found the parasite in another case.

Jackson (*Boston Med. and Surg. Journ.*, November 30, 1905) insists on the difficulty of diagnosis in **malignant disease of the lung or pleura,** and gives details of ten cases, four of primary growth, six of growth secondary to disease elsewhere, which illustrate the point. The difficulty of course is much less in the second class, but even in that there is often a complete absence of conclusive symptoms.

He lays especial emphasis on the absence, as apt to mislead, in these cases, of the cachexia so characteristic of malignant disease in general : this he attributes to the fact that the growth does not interfere with nutrition in the same way as in abdominal cancer, while a vital organ being the seat of growth, life cannot be prolonged, as in many cases of external cancer. This absence of cachexia was a marked feature in all the cases he reports.

In the physical signs of malignant growth of the lung or pleura, there is often nothing to be made out, which is pathognomonic ; no sign of a massive tumour, no pressure symptoms. Pleural effusion often occurs, whether the growth is primary in the lung or in the pleura, and there may be nothing to suggest any diagnosis other than acute pleurisy. In one of the reported cases, the illness dated from a chill caught when driving in a storm three weeks before entering hospital. In another, there was the same acute, or sub-acute, onset, and the only circumstance up to the end, which could lead to a suspicion of malignant disease, was the rapid re-accumulation of the pleural effusion necessitating repeated tapping. In both these cases the disease ran its course in a few weeks.

Where the growth is disseminated throughout the lung, the signs found on examination are the ordinary ones of consolidation; bronchial breathing, increase of vocal fremitus and resonance, crepitations. The confusion is increased by the fever which is frequently present, and to which the author calls special attention, the temperature, for instance, in one of his cases, rising occasionally to 104°. The condition then is very apt to be taken for some acute inflammatory disease, such as pneumonia, or, if the signs are apical, as in one of these cases, tuberculosis.

Pfahler (*Amer. Med.*, February 10, 1906) gives the results of the **treatment of mediastinal carcinoma by the Röntgen rays** in six cases, the mediastinal growth in all being secondary to carcinoma of the breast. Three of the patients are either well or nearly so, three died, but in all there was improvement, if only for a time. This result may not seem very encouraging, but even so, this line of treatment offers more hope than can be expected from any other means. In only one case was there any dermatitis, and that only slight, justifying the author's claim that he has shown that the Röntgen rays can affect deep-seated disease without destroying the superficial tissues.

The signs, on which the diagnosis of mediastinal growth was made, were dulness, or sometimes prominence over the sternum and in the intercostal spaces in its neighbourhood, and the symptoms, cough, pain, dyspnœa, asthmatic attacks, and difficulty of swallowing. The diagnosis, in some cases, was verified by radiography. Under treatment, there was in nearly every instance increased comfort, with lessened cough and pain, and, in some, diminution in the area of dulness could be made out. In one or two cases, this shrinking in the mediastinal mass is reported to have been confirmed by radiography.

Certainly, by this treatment, the evil day seems to have been postponed for a time at any rate, and the patients given a period of activity, and even power to work. The paper contains one striking instance of great relief from severe neuralgia, and loss of power in the arm and hand, due to deep pressure on the cervical plexus.

Lawson and Struthers Stewart ("A Study of Some Points in Relation to the Administration of Tuberculin," *Lancet*, December 1905) have been testing the applicability of Wright's discoveries, the theory of which has been explained in a previous number of THE PRACTITIONER, to the treatment of pulmonary tuberculosis, and in this article contribute a preliminary communication on their results. As regards the tuberculo-opsonic index of healthy people, they are able, adding the series published by Bulloch, to present 91 cases with an average of 0·95. Their tubercular cases fall into two groups, cases of apparent recovery, of which they studied 50, and 25 cases still undergoing treatment, *i.e.*, of still active but, in no instance, acute disease. The former are defined as having fulfilled all known tests of cure, and could now be regarded as useful lives. In ten of these the index was below 0·9 (in two as low as 0·5, in one 0·6) and in six above 1·2. The average index of 25 still under treatment was 0·7.

The conclusion drawn is that an index above 1·2 or below 0·9 is very suggestive of tuberculosis.

The effects of the inoculation of small doses of tuberculin on the three classes was as follows:—In the healthy an immediate rise in the index, in the tubercular a primary fall (negative phase) of variable duration followed by a rise. In no instance apparently was this negative phase absent, though in some re-inoculation was needed to demonstrate it.

The varying length of the negative phase, five per cent. of the cases being still in this position at the end of a fortnight, renders a daily observation of the index absolutely necessary, if the best result is to be obtained from the use of tuberculin.

Such repeated examinations would of course be excessively laborious, and the suggestion that the temperature curve might take the place of the blood examination was considered, but found to be valueless. In only four out of the 25 active cases was there any temperature disturbance, and this again bore no relation to the length of the negative phase.

THE CAUSES OF PRURITUS ANI.

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PRURITUS ANI is without doubt one of the most troublesome affections met with in rectal surgery; it has become almost a byword for unsuccessful treatment, and, for many years, has been one of the happy hunting grounds of quacks and charlatans. The treatment of this condition is most difficult and frequently unsatisfactory. Patients go from one doctor to another, and from one surgeon to another, seeking relief and often getting only temporary amelioration of their trouble. This is all the more disappointing, because the patient's complaint is a simple one. He has itching of the anus, and only wants something done to stop it; the very simplicity of the request only renders failure of the treatment the more annoying.

The real reason for this unsatisfactory state of affairs is that we know very little about the true causes of the condition. The different causes for pruritus that have been described would fill a volume, many of them are merely the results of the condition, many have no relation at all to it, and again many are predisposing, or accessory, rather than true causes, and so can help us but little in the treatment.

Pruritus ani is no more a disease than is headache, or vomiting, but, like these, is a symptom, which may complicate many diseased states, and it has only earned the distinction of sometimes being called a disease because, in many cases, we have failed to find the true cause. Pruritus ani has no pathological existence, and the only lesions associated with it are the results of scratching, or of the condition which gave rise to it. In cases of old standing, there is almost invariably a characteristic change in the anal skin. The skin is thickened and warty, or has a washleather-like appearance, but these changes are purely the result of scratching, or of the damp condition of the parts, which is so frequently associated with pruritus ani.

I am convinced that the exciting cause of pruritus is a local one in all cases, and that constitutional states have merely a predisposing or secondary influence in causing the itching. It must be admitted that, in some cases, the most careful search fails to discover any local cause, but this is because our knowledge is insufficient to enable us to discover it, or perhaps to recognise it when seen.

The anal skin and mucous membrane have certain peculiarities, which have an important bearing upon the cause of pruritus. The mucous membrane lining the anal canal and the skin at the anal margin are richly supplied with sensory nerves, which act as the sentinel of the rectum, and these nerves are some of the most sensitive in the body to tactile influences. The skin and mucous membrane within the grip of the sphincters is kept in constant forcible contact each part with the other. The sensation of itching may commence at any point of the anal canal, or the skin surrounding the anal orifice; as a rule, however, the most intense sensation is at the muco-cutaneous junction, which is also the point at which the largest number of nerve endings are congregated. Any source of irritation within the anal canal, or at either end of it, may be the cause of the itching, and one of the first things to be done, in examining a patient suffering from pruritus, is to thoroughly search this area.

The external parts should first be examined, but one must not be misled into thinking that the obviously abnormal condition of the perianal skin is the cause of the itching. On the other hand, it is invariably the result of scratching, or of applications which have been made to relieve the irritation. The anal canal itself must be most carefully examined, first with the finger, and then with a small anal speculum. Every portion of the mucous membrane should be examined, and it is important to carefully examine each of the little anal pockets, or valves of Valsalva, which are to be seen just above the muco-cutaneous junction. Sometimes a minute ulcer is found at the bottom of one of these pockets, or a small fistulous tract opening into one of them, is discovered. The best way of examining these valve pockets is with a small bent probe. I have seen several cases in which pruritus had resulted from a small fistulous tract at the bottom of one of these valves, and which was cured by division of the valve and laying open of the fistulous tract. After the anal canal has been thoroughly searched, a short proctoscope tube should be introduced, and the upper portion of the anal canal and the lower portion of the rectum examined, and lastly the rectum itself should be examined, up to and above the recto-sigmoidal junction, with the electric sigmoidoscope. Of the lesions in the anal canal, which may cause pruritis, the most common are, fissure, a submucous fistula, ulceration in one of the anal valves mentioned above, and small polypi.

One of the common causes of pruritus is a constantly damp condition of the anal skin ; this is due to some leakage, which allows the rectal mucus to escape, and keeps the skin, at the anal margin, in a constantly damp condition. The causes of this leakage are numerous. Internal piles, or small polypi, by partially prolapsing into the sphincter, cause mucus leakage, and, I believe, that this is the usual manner in which piles cause pruritus. Fissure, fistulæ, partial procedentia, worms, and several other conditions may be the cause of pruritus by causing leakage in a similar manner.

That leakage of moisture from the anus may cause pruritus is, I think, proved by the fact that a certain degree of pruritus is not uncommonly complained of by patients during the healing stage after an operation for fistula, in which a certain amount of leakage is inevitable.

Of the causes within the rectum itself, which may give rise to pruritus, one of the commonest is catarrhal proctitis. This is a cause, which is frequently overlooked owing to the surgeon being unacquainted with the proper methods of examining the rectum, or owing to his not having the proper instruments at his disposal. The commonest form of proctitis, which I have observed in association with pruritus, is the hypertrophic catarrhal form. This is characterised by hypertrophy and œdema of the mucous membrane, accompanied by an excessive secretion of acid mucus. There is another form of proctitis, in which the mucous membrane appears congested and granular on the surface. Both these forms of proctitis are very chronic, and are usually quite unsuspected, until an instrumental examination of the rectum is made ; in both of them there is an excessive secretion of acid mucus, and it is the constant presence of this irritant material in contact with the sensitive mucous membrane of the anal canal, which causes the itching.

I have seen several cases, in which the cause of the pruritus could be definitely traced to the accumulation of this irritating mucus in the upper part of the anal canal. In one case, the patient had, himself, discovered that this was the cause of the irritation, and was in the habit of stopping the itching by removing the mucus from his rectum.

On examining this patient with the sigmoidoscope, I found that he had typical hypertrophic catarrhal proctitis, and, when this was cured by suitable injections, the pruritus ceased.

In many cases of pruritus, there is a very definite history of gout or of chronic dyspepsia, or the condition is very definitely associated with the taking of alcohol, or of some special form of diet. I believe that, in these cases, the pruritus results from a proctitis set up by the alimentary or constitutional disturbance, or is due to the products of abnormal fermentative processes, accumulating in the lower portion of the rectum.

There are some cases of pruritus ani associated with a special tendency to perspiration around the anus, and between the buttocks, and it would seem as if the pruritus, in some of these cases, resulted from the damp condition of the skin induced by the constant perspiration. At any rate, attempts to keep the parts dry and prevent perspiration seem to alleviate the itching.

Although the warty and rugose condition of the anal skin is the result of the pruritus rather than its cause, there is no doubt that, in old standing cases, the fissures, which form between the ridges on the skin, have a great deal to do with keeping up the irritation, consequently, attention must be paid to getting the perianal skin back to its normal condition. This is best done by painting it occasionally with carbolic acid, and the frequent application of weak carbolic fomentations.

I should like finally to insist that there is always a local cause for pruritus ani, and especially to insist upon the importance of a most thorough examination, not only of the anal canal, but also of the rectum itself in all cases.

I feel sure that it is only by ascertaining the real cause of the condition in each case, and by then treating that cause by scientific and common-sense methods, that we shall be able to remove the cloud that now rests over the treatment of pruritus ani, and to rescue it from the hands of the quacks and the patent medicine vendors.

UTERINE CURETTAGE.

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UTERINE curettage is a simple operation in itself, but it is one which is too often advised and carried out on mere symptoms. It is very important to go further than mere symptoms, and to consider the pathology of the endometrium before recommending curettage.

No one can doubt that the simple operation will cure certain conditions of the endometrium, and, as an aid to diagnosis of intra-uterine growths, curetting is of the greatest value, and few of the more important points may well be discussed.

First, let us take the symptoms which may lead to a suggestion of curettage.

1. *Leucorrhœa.*—The patient complains of more or less white discharge of a watery consistency. When the uterine glands are irritated and congested, they secrete a thin watery fluid, which picks up a certain amount of mucus from the cervical glands, and this mucus is turned white when it meets the acid vaginal secretion.

Now what is the cause of this excess of fluid discharge from the uterus? The cause is generally congestion; the mucous membrane is œdematous and vascular; there is no active invasion of bacteria. In other words, there is no real active endometritis present. A displacement, or chronic subinvolution, will lead to this condition, the uterus feels bulky, there is venous and lymphatic stasis. If the uterus is replaced, and the unhealthy mucous membrane removed, the congestion will disappear, and healthy mucous membrane will be regenerated. The symptoms, in a great many cases, date from getting up after labour. If this condition of the endometrium must be called endometritis, I should suggest calling it catarrhal or congestive endometritis, because, as a rule, no certain pathological alterations can be found in the endometrium removed in cases of chronic leucorrhœa. Thus when congestion is due to recent pregnancy, or a displacement, removal of the œdematous mucous membrane will be followed by complete relief to the patient.

2. *Yellow Discharge.*—A yellow discharge means pus, and here we have an entirely different pathological condition of the endometrium. The endometrium is infected with bacteria, and true endometritis exists. This condition may be acute or chronic. In the acute form, the infection is so extensive, spreading from the uterus to the tubes and the peritoneum, that curetting is rarely if ever thought of, and ought certainly not to be done in any circumstances. We will discuss the use of curetting after abortion later on. Gonorrhœa, in its less acute form, is perhaps the most common cause of yellow discharge, and we must not forget tubercle. Hæmorrhage is a frequent additional symptom in the latter case.

Opinions differ as to whether it is right or wrong to curette a uterus discharging pus. Personally, I should strongly advise against it. We are dealing with a septic uterus, and a sharp curette opens up new roads to infection, by removing a layer of protecting leucocytes, laying bare lymphatics, lighting up latent mischief in the tubes, or even causing a direct spread of infection to these parts. I have more than once seen curettage of the uterus followed a few months later by bilateral pyosalpinx.

Therefore, in cases of chronic yellow discharge, it is advisable not to curette, but to be content with dilatation and antiseptic swabbing, followed by drainage of the uterine cavity. Curettage of the infected cervical canal will do no harm, because the natural drainage is good and the cervical tissues are much less vulnerable to bacterial invasion than is the delicate endometrium which lines the uterine cavity. The uterine cavity is indeed a very natural incubation tube.

Uterine Hæmorrhage.—There are two forms of uterine hæmorrhage :—

(*a*) Menorrhagia : exaggerated physiological bleeding ;
(*b*) Metrorrhagia : bleeding, not in distinct relation with the menstrual flow.

(*a*) Menorrhagia.—The endometrium may be primarily at fault, that is, the bleeding may be due to congestion, new growth (benign adenoma) or tubercle ; or the endometrium may be secondarily affected, being then altered by some other cause, such as a fibroid tumour, or arterial degeneration.

Now, if we are dealing with primary affection of the mucous membrane, such as congested endometrium, or a benign overgrowth of the glandular elements, the curette will effect a cure.

One often finds that the interior of the uterus contains numerous polypoid tongues, rather like adenoids of the nasopharynx, to which the name polypoid or villous endometritis has been given. Curettage will not cure tuberculosis, and, if the curettings show tubercle, hysterectomy is advisable. I think that perhaps, in tuberculous disease, metrorrhagia is more likely to be the symptom.

If, however, the menorrhagia is caused by a fibroid growing in the walls of the uterus, the mucous membrane is not primarily at fault. The whole menstruating area is much increased, and one frequently finds no great alteration in the endometrium beyond an œdematous condition of the cells. Therefore intramural fibroids cause menorrhagia by enlarging the uterine cavity, and, if the uterine cavity is not enlarged there is rarely excessive menorrhagia. The increased blood supply, associated with a large fibromyoma, must also help to cause increased flow of blood at the period.

Curetting for menorrhagia with fibromyoma is a time-honoured palliative method of tiding a patient over the menopause. But, since we do not remove the primary cause, the hæmorrhage recurs after a varying period of relief. It is impossible to predict the date of the menopause in a patient with a normal uterus, much less so in a patient with an intramural fibroid; these tumours frequently place the menopause six or seven years further on in life. For this reason, I do not think that we must rely too much on curetting for fibroids, and, further, there is a possibility of infecting the fibroid by curettage. One becomes more and more impressed with the fact that the dangers of fibroids are by no means over when the menopause is established, because the very atrophy that is looked for as a cure may be the cause of necrosis, septic or otherwise.

Now let us turn to the group of cases with arterial degeneration. They are met with in chronic nephritis, but may be present as an independent local condition. The intramural branches of the uterine artery are in a marked state of arterio-sclerosis, the muscle is less striated than normal, and there is a considerable amount of fibrous tissue thrown out between the muscle bundles. This condition is called chronic myometritis. Menorrhagia, and sometimes metrorrhagia, chronic pain, a bulky uterus, are the signs and symptoms. Curetting is the routine treatment, and the operation may have to be repeated several times. Each operation may relieve for a few periods, and then there is a relapse, with perhaps almost constant bleeding. I do not know that this condition can be diagnosed except by the failure of curretting to relieve it. The failure is due to the fact that the primary cause, sooner or later, builds up a faulty endometrium. Hysterectomy will then have to be considered.

In cases with albuminuria, or a low specific gravity of the urine, curetting may do good as a temporary measure, if the profound anæmia is thought to be due to the hæmorrhage; but it is perhaps wiser to try ergot, or other palliative drugs, rather than any form of operation in chronic Bright's disease with a so-called interstitial endometritis.

(*b*) Metrorrhagia.—The commonest causes of uterine hæmorrhage are those connected with early pregnancy. We must be careful not to mistake this uterine hæmorrhage for tubal abortion, or ruptured ectopic gestation. This mistake has been made more than once. In these cases one or two periods may be missed: the patient then complains of pain followed by flooding. A decidual cast may be passed which the patient mistakes for an ovum. If one sees a patient soon after the symptoms started, there may be great difficulty in diagnosing a hæmatocele, because the retro-uterine pouch may be full of liquid blood, and an extra-uterine condition possibly overlooked. One should never curette for acute hæmorrhage, and the blanching of the patient and the great pain should put one on the track that there is more hæmorrhage than is apparent. Later on, the diagnosis is easy because the hæmatocele will then be firmly encysted.

In cases of incomplete abortion causing hæmorrhage after the third month, and in recent abortion, the blunt curette should not be used to get rid of retained bits. The cervix is nearly always sufficiently patulous for the introduction of a finger, and the finger is the best blunt curette for loosening the retained products. In very early abortion of one or two months, there may not be enough dilatation of the cervix; the blunt curette can then be passed in order to break up the placental remains, but it must be used with great care, for the uterine wall is so soft that the curette may perforate it easily. Thus, on the whole, I think ovum forceps would answer better, if used with great caution.

It frequently happens that a patient continues to bleed for several weeks after an abortion. The uterus may be only slightly enlarged; there is, in fact, a decidual endometritis. Curetting will at once cure this condition. One may, in fact, use a blunt curette after 10 days or a fortnight, if the cervix has closed down. The curettings should be examined microscopically, because the possibility of chorion-epithelioma as the cause of the hæmorrhage must be considered.

There is a great difference of opinion as to whether it is wise to curette a septic uterus following on labour or abortion. Personally, I should strongly advise against such a proceeding. Certainly only a blunt curette should be used, and you cannot rely on this instrument to remove retained products. The safest treatment to adopt is to dilate the os, if not already dilated enough to admit the finger. The whole cavity of the uterus should be explored in this way, and the septic necrotic retained products removed. The cavity is then thoroughly washed out with a copious douche of hyd. perchl., followed by saline solution and drainage. If the curette is used, venous sinuses and lymphatics are reopened, and the protecting barrier of leucocytes is interfered with, and possibly removed in places, and, further, the Fallopian tubes may become infected.

We must clear out the retained products, and the blunt curette is not the instrument to use for this purpose. We are working in the dark, and we may perforate the uterus. Again, after full-term labour, the placental site is not known, so that the whole of the uterine cavity must be gone over with the curette. This is impossible in a uterus of any great size.

The Use of the Curette as a Means of Diagnosis.—The blunt curette is of great service as a means of diagnosis in cases of bleeding at the menopause, or after, or even before, for I have myself seen two cases of carcinoma of the fundus uteri, at 34 and 50 years. If we take a case of bleeding after the completion of the menopause, the uterus may not even be enlarged, and our bimanual examination may reveal nothing. How are we to find out the condition of the endometrium?

Most patients, suffering from early carcinoma of the body of the uterus, are either single or sterile. It is a matter of great difficulty to dilate the cervix sufficiently to admit the finger, and the cervix may even be ruptured in the attempt. The cervix must either be split anteriorly, or we must use the curette to snare a bit of mucous membrane. The cervix will only require a little dilatation, and the curette will bring away small pieces of the friable growth, if it exists. The naked-eye appearance of these pieces of growth is almost typical in every case. Good large pieces, which are brittle and whitish in appearance, may come away, and, contrasted with the gelatinous aspect of the benign mucous membrane, will often settle the diagnosis without the help of the microscope.

Again, if profuse hæmorrhage occurs on manipulation with the curette, this nearly always means malignancy. The microscope will at once clear up any doubt that may exist with regard to the curettings. We can thus, in older patients, arrive at a correct diagnosis by simply passing the curette into the uterus, and removing a small portion of the endometrium. Again, if profuse hæmorrhage continues after abortion or hydatid mole, the blunt curette will bring away small pieces, which, on examination, may decide the question of chorion-epithelioma. All curettings should be examined under the microscope as a routine practice, in order to note any peculiar pathological condition. Thus tubercle, carcinoma, chorion-epithelioma, decidual endometritis, etc., will be recognised.

When the uterus is the cause of sterility, a condition so often associated with dysmenorrhœa, it is always best to curette the uterine cavity after having dilated the cervix. A fresh mucous membrane is more likely to accept a fertilised ovum. Again, in patients who frequently abort, the removal of the diseased endometrium is indicated.

In the treatment of inoperable carcinoma of the cervix, where septic absorption is one of the common causes of immediate distress, the curette is also of great service. Curetting the fungating mass, and subsequent treatment of the raw surface with strong formalin, frequently does away with sepsis, hæmorrhage, and pain. I have seen a foul malignant growth of the cervix transformed into a clean, dry, painless induration, and the general condition of the patient greatly improved by this method of treatment.

The technique of the operation is simple enough. I always use metal dilators, and, if they are carefully inserted in the direction of the canal without great force, there is not much danger attending their use. The introduction of laminaria tents has the advantage of producing a more gradual dilatation, but this means that the patient has 16–24 hours' pain before the operation, which is highly objectionable.

A sharp curette is the best instrument for ordinary cases, not connected with pregnancy or abortion, but if a pregnancy or an abortion has occurred within a month, the blunt instrument should be used. Further, in curetting for diagnostic purposes, the blunt instrument is preferable.

A blunt flushing curette is a very useful tool to use after curettage with the sharp instrument.

I have not gone into the details and dangers of the actual operation, because it is beyond the scope of this short paper.

ON SOME AFFECTIONS OF GASTRO-INTESTINAL TRACT IN GOUTY SUBJECTS.

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THAT gouty patients are more subject to dyspeptic ailments than other people, and that these conditions are sometimes worse before an attack of gout, have been matters of common knowledge for generations. Sydenham has mentioned them, giving his personal experiences. It is, of course, probable that many cases of so-called gouty dyspepsia are quite independent of gout, and are due to the many other causes which produce indigestion.

In some gouty people the symptoms are, however, much more severe than a simple dyspepsia, and there may at times be retching with the bringing up of mucus, or actual vomiting. Dysphagia is also a symptom complained of, and a very sensitive condition of the epigastrium associated with flatulence.

Less frequently, the symptoms become more acute, and a patient, who perhaps for years has been only subject to slight "indigestion," may suddenly get an alarming abdominal illness with severe pain, retching, and extreme tenderness in the upper part of the abdomen, spreading thence over the whole.

I admit that, at one time, I was not a believer in these severe attacks as being due to the same cause as gout as manifested in the joints, but a more extended experience has convinced me that they are part and parcel of the disease. Of course many of the older writings describing gout of the stomach, in all probability, include records of cardiac disease, angina, aneurysm, renal conditions, gall stone, etc., etc.

Our knowledge of the causation of gout is still defective; at the same time we have altered our views in the last few years, and few medical men now look upon excess of uric acid as being more than a by-product of some alteration of metabolism. Of the cause and the nature of these changes we know little. Recent suggestions have drawn our attention to the rôle of the gastro-intestinal tract, but whether it stands in a primary or secondary relation to the actual chemical processes at the base of the disease, is still a doubtful point. It is quite as probable that many of these gastro-intestinal conditions are due to an alteration of the intestinal secretions from an internal cause, as that they are due to bacteria and the formation of toxins in the intestines, or to some alteration of the epithelial cells, so that they take up chemical compounds of a different nature from the ordinary. Although many conditions of the joints may be induced by a definite bacterial cause, I do not think there is as yet sufficient evidence that there is anything of a specific bacterial nature, in the alimentary canal, giving rise to toxins which produce the gouty state.

Looking at the matter in widest manner, and remembering the hereditary element in the disease, I do not consider it as being at all probable that the solution of the subject will ever be found in any special micro-organism. The explanation is, to my mind, only to be found in a closer and more extended study of that most difficult subject, the actual chemistry of the cell protoplasm.

That toxic substances may be excreted with the bile is probable in the cases of burns, in which a secondary ulceration of the duodenum sometimes occurs. This, at any rate, is more easy to understand than a reflex nervous explanation. I have notes of several cases in which, in gouty people, the so-called indigestion was much more referable to the duodenum than to the stomach. If, however, the severe intestinal attacks were always primary, would one not expect the joints to be more often worse after the attack? This, of course, may occur at times, but especially in patients after middle age, one often finds that the gastro-intestinal illness takes the place of an attack of articular gout, and there is after it a large excretion of uric acid. In these attacks, I feel sure that the gastritis is usually secondary to a duodenitis. There seems to be an attempt to get rid of some poison, and frequent, and at times alarming, retching, with the bringing up only of mucus, takes the place of actual vomiting. These bad attacks come on suddenly, and usually there is no immediate cause to be found. There is sometimes a little diarrhœa at the onset, and the stools may have a peculiar odour, but, during the height of the attack, there is usually constipation.

It is probable that these attacks run a definite course, whatever drug be used, and so further resemble acute articular inflammations. Haig[5] recommends salicylate, and Mercer[6] colchicum. A question of interest is whether there may not be, in some of these acute attacks, a vascular condition similar to what probably occurs in migraine; perhaps a contraction of the blood vessels, followed later by a dilatation. I have recently had a case of attacks of polyuria, in a man of about 40, who formerly suffered severely from migraine. Recently the migraine had quite disappeared; but, in the last six months, he had been subject to sudden attacks of passing large quantities of urine. Most of the time these lasted (a few hours to a day) he felt generally ill, and on the occasion on which I saw him, he had actually fainted. There was no albumin present, and the organs of the abdomen and of the chest were quite healthy. The condition can be best explained on the theory of a sudden dilatation of the vessels of the kidney.

We know very little about the vaso-motor supply to the vessels of the digestive tract. It is consequently only a suggestion to say that some of the severe gastro-intestinal attacks may be associated with some vascular state analogous to what occurs in migraine, or in the case mentioned. I have, however, seen, in a patient the subject of hereditary gout, and one who used to suffer from migraine, the formation of an indefinite swelling in the duodenal region, which suggested something of the nature indicated. This occurred during two severe attacks, and entirely disappeared after each. Of course, it may have been due to some extension of a general inflammation. There was no sign of ulceration, and, between the attacks, the patient was in his usual health. The liver, gall-bladder, and kidneys were apparently normal.

An atrophic condition of the lower part of the intestine is often met with in the subjects of the gouty diathesis, who suffer from chronic intestinal trouble. It is usually found in old people, and is a condition difficult to influence in any way. I think it is quite probable that, in these people, the atrophic state affects the whole of the intestinal tract. Is it possible that it is of the same nature as the chronic interstitial nephritis of gout? Allen[1] suggests that, in the gradual development of gouty kidney, a few glomeruli get put out of action every now and then, by means of the formation, here and there, of small microscopical areas of scar tissue. In this way he says that one can account for the gradual rise of pressure in the remaining tubules, giving rise to a larger quantity of water passed, but, as the parts through which the filtration and secretion take place are still healthy, there is as yet no albumin in the urine. De Grandmaison[3] mentions that in cases, at about this stage, it needs but little alteration in the health to produce albumin. I think that, in chronic cases, it is quite probable that a similar fibrosis and atrophy occur in the intestinal tract, and due to the same gouty irritant.

Attacks of intestinal gout, in middle-aged people, often simulate appendicitis. Haig[5] mentions a case which came on after calomel, and another, also resembling appendicitis, in which the temperature rose to 100°, and in which there was very severe pain in the right iliac fossa. Mercer[6] also mentions a case of intestinal gout, in which there was severe pain in the epigastrium, extending to the right iliac region, with frequent vomiting, dysphagia, and constipation. After the attack, which lasted for about two days in a severe form, much uric acid was passed.

It is interesting to note that, in seven cases of gastro-intestinal gout, mentioned by Grube,[4] in three the HCl of the gastric juice was diminished, and in four it was absent. In three cases lactic acid was present, and in two there was atony of the stomach with lessened motility.

Fatty diet is never suitable for patients who are subject to intestinal trouble, they do not seem able to digest it, and it gives rise to flatulence; perhaps the odour of the stools, which I think was first noted by Grube,[4] may be due to the fatty acids present. Mucus in the stools has also been noted; in the best example, which I have come across, it was associated with atrophy of the intestinal mucous membrane.

Pruritus ani is also one of the conditions associated with intestinal dyspepsia in gouty people. In one case, which I have treated, although the irritation was worse at night, it also often came on after a meal. Allen[1] has written about the association of duodenal complaints with the nervous system, and with skin affection such as urticaria, eczema, and pruritus.

The following case is an example of an acute gastro-intestinal attack in a patient, aged 51. In the past, he had suffered from many attacks of subacute articular gout in the hands and feet, and had for years been subject to migraine. During the last few years the latter had been much better, and, in the previous twelve months, the hands had greatly improved, and the feet had not troubled him at all. The first and second intestinal attacks had been slight, the third severe, and the fourth, about 18 months after the third, quite alarming. I only saw him during the third and the fourth. There was no immediate cause to be found for either, but, before both, there had been prolonged mental overwork, and, on each occasion, he was about to go for a holiday. In fact, the last attack came on the very morning of the day he had settled to start, the third on the day before he had arranged to leave town. The third attack resembled appendicitis, as the pain, after starting in the stomach area, gradually travelled down the right side to the iliac fossa, and there was much pain on pressure here, but the left umbilical region was also tender with a feeling of some indefinite swelling. The temperature was never above 101°. This attack was only severe for about two days, and the symptoms went off so quickly that, in about a week, the patient was as well as usual. There was constipation for a couple of days, but, after this, the bowels were opened naturally.

The fourth attack came on about a year and a half later. In the interval the health had been good, and he had not found it necessary to stay indoors for a single day, and, as already mentioned, the gouty condition was apparently better, and the fingers less swollen than I had ever seen them. He felt almost as well as usual the day before. The attack began about 2 o'clock in the morning, and got worse during the day. There were in the beginning two rather liquid stools. During the day, there was a good deal of retching, with the bringing up of mucus only. The pain was at first in the epigastric region, but soon spread down the right side to the iliac fossa. There was a good deal of flatulence. On the second day, the condition was quite alarming, and, had I not seen him before in a similar attack, I should have recommended an exploratory operation. On this second afternoon, the abdomen was distended, the whole very tender, the worst part being that to the right of the umbilicus. The liver dulness was diminished. There was no dulness to percussion in any part. There was severe pain all down the right side. Since the preliminary opening of the bowels, nothing had been passed, and there was difficulty in passing water, on account of the condition of the abdomen. The retching in the afternoon was very severe.

On the third day, the symptoms were much better, and the flatulence was less, as it had been relieved by enemata, rectal tube, etc. On the fourth day, it was possible to thoroughly palpate the abdomen, and there was a large indefinite swelling to be felt to the right of the umbilicus. This was thought at first to be fæcal, but no results of enemata confirmed this view, and from the way in which it gradually disappeared, I am inclined to think that it was of an inflammatory nature. After about a fortnight, it had quite disappeared. The temperature, on two occasions, was between 102° and 103°. Liquid food could be taken throughout in small quantity, but there was the greatest difficulty in swallowing. As the attack went off, there was a condition of stomatitis and pharyngitis, which, for a couple of days, gave a good deal of inconvenience. For most of the time the tongue was bad : it was reddish in parts, and in other parts coated. It was also somewhat swollen and fissured. Grube[4] also found this condition of tongue in cases of this kind. The mouth was very dry, but the most prominent feature was the dysphagia, which continued into the period of convalescence. I could not make out any enlargement of the liver after the attack, and there was then no albumin in the urine. About 10 days after the commencement of the illness, the prostate got inflamed, and there was, for a few days, some trouble in passing water. After the attack, the urine contained a large amount of uric acid.

Stockton[8] says that the dyspepsia of gouty patients is associated with hyperacidity, but I do not know of any notes of cases in which chemical examination of the stomach contents proved this to be the case. He also says that the hyperchlorhydia does not respond to the usual remedies. The question is whether there is hyperacidity in these attacks.

In the cases mentioned by Grube,[4] in four out of seven, HCl was absent, and, in the remaining three, it was diminished.

I think we can divide the varieties of gastro-enteritis in the gouty into two divisions, the chronic and the acute. With the former we are all familiar--dyspepsia, duodenal catarrh, atrophic conditions of the bowel, etc.

The acute condition is usually seen in patients who have suffered from gouty joints, and often from migraine, and who develop these acute intestinal conditions about middle life, sometimes after their articular gout has improved, and they imagine that they are getting the better of their old enemy.

Grube classifies them according to whether there is—

1. No marked chemical alteration of the contents of the stomach ; or
2. Definite chemical change.

The former would probably correspond with the chronic cases, the latter with the acute.

The question still is : Are these attacks primary, and the actual cause of the development and increase of the gouty condition, or, are they to be regarded as secondary, and due to the general alteration of metabolism acting on the digestive organs ?

Stockton[6] mentions that gastro-intestinal cases are associated with enlargement of the liver, and are likely to go on, if not treated, to acute attacks of the joints : so he apparently looks on them as primary. On the other hand, he mentions that gouty people suffer from gastralgia, independent of diet, and so perhaps regards this condition as a secondary one. Minkowski[7] mentions the possibility of the digestive organs acting as the doorway for the entrance of the gouty element ; his experiments with adenin, however, rather favour the other view, which is, that the inflammation can be produced from within. Adenin, if injected subcutaneously in rabbits, will produce an excess of uric acid in the urine, and, at the same time, cause an inflammation of the gastro-intestinal tract. And there are many drugs which can produce inflammation of the stomach and duodenum, although they may not be given by mouth.

So far as one has seen, clinically, it is usually very difficult to find an actual cause for either the chronic or the acute conditions. Surely in acute cases, such as the one mentioned, the irritation must be due to the same cause as, at another time, will give rise to an attack of articular gout. The inability to digest fatty substances, the nature of the stools as mentioned, and the duodenal nature of the catarrh make one wonder if the liver is not directly at the root of the mischief in all cases. If the irritant chemical substance from the liver does not get into the general circulation and affect the joints, it will come out into the alimentary canal.

In the absence of any definite proof, and in the present state of our knowledge, any theories on this subject must be of a very speculative nature. I think, however, that further study of these gastro-intestinal conditions, whenever the opportunity is afforded, would add materially to our conceptions of the disorder of metabolism, which we know as gout.

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Reviews of Books.

The Nursling. The Feeding and Hygiene of Premature and Full-term Infants. By Pierre Budin, Professor of Obstetrics, University of Paris; Director of the Clinique Tarnier, Member of the Academy of Medicine. Authorised translation by Wm. J. Maloney, M.B., Ch.B., Fellow of the Obstetrical Society of Edinburgh; Ettles Scholar, etc. With an Introduction by Sir Alexander R. Simpson, M.D., LL.D., D.Sc., Emeritus Professor of Midwifery and Diseases of Women and Children, University of Edinburgh. 111 Diagrams in colour and other Illustrations. Pp. xxiv + 199. London: The Caxton Publishing Company.

The names on the title page of this handsome volume are quite sufficient to commend it to the members of the medical profession, and to all interested in the preservation of infant life. Indeed, its publication, at a time when the questions of a decreasing birth-rate and excessive infant mortality are agitating the public mind, is exceedingly opportune. The book consists of ten lectures, delivered to his students by Professor Budin, in which he explains the methods which he has found successful for preserving the life and promoting the growth of the newly-born, and which are, to a great extent, a detailed account of cases, illustrative of his work at the Consultations for Nurslings, at the Maternité, and at the Tarnier Clinique. The lectures themselves are particularly lucid and instructive, and form exceedingly delightful reading; but they are rendered doubly instructive by the very clear and beautiful graphs by which the text is illustrated. These graphs at once convey to the mind of the reader, far more effectively than any words could do, proofs of the great value of Professor Budin's system. That his system has proved to be signally successful is abundantly shown by the statistics which are scattered over almost every page. The first four lectures deal with the cases of children, prematurely born, and, from various causes, the abnormally weak; the last six with the cases of full-term infants. Throughout the whole, as might be expected, the utmost attention is given to the consideration of diet, and the greatest stress is laid upon the pre-eminent value of breast feeding. We draw especial attention to this, as, for various reasons, into which we need not enter, the artificial feeding of infants is so much resorted to in the present day. Where such feeding becomes a necessity, it appears that the use of sterilised milk has met with unqualified success. The question as to whether sterilised or refrigerated milk is the better food for infants is just now one that is under the consideration of some of our leading physicians, and some have not hesitated to express themselves in favour of the latter. At the same time, it is urged that arrangements are now being made at most of the municipal depôts for the periodic weighing, under medical inspection, of all the infants for whom sterilised milk has been supplied. We venture to think that a vast amount of testimony in favour of the use of sterilised milk is already to hand in Professor Budin's lectures.

Other means for the preservation of infant life, to which detailed reference cannot, of course, be made here, are ably dealt with in this volume. It will doubtless be sufficient to express our unqualified approval of the work before us, and to congratulate Mr. Maloney on having proved himself to be so efficient, and so sympathetic a translator.

Infant Education. By ERIC PRITCHARD, M.A., M.D. (Oxon.), M.R.C.P. (Lond.). Consulting Physician to the Barnet Home Hospital for Children; Senior Physician to the St. Marylebone General Dispensary; late Clinical Assistant at the Victoria Hospital for Sick Children, etc. Pp. xiii + 190. London: Henry Kimpton. 2s. net.

AT first sight, the title of this little book might be considered a misnomer, but Dr. Pritchard has used the word "education" in its classical and most accurate sense, and wishes us to understand that his object is to show how to develop, to render fit for the performance of their various functions, the internal organs, the muscular systems, and the senses of the future generation. This, of course, includes the preservation and well-being of infant life in the best possible way.

It is most interesting to know that, in the borough of St Marylebone, there is a Health Society, which has for its aims the prevention of the spread of tuberculosis, and the reduction of infant mortality among the poor, residing within the area of its operations. In order to effect the latter, this Society has an army of wardens, nurses, and visitors, whose duty it is to advise the mothers as to the management of their babies, and on matters relating to their own health, and the system is founded to a great extent upon the lines of the "Consultations de Nourrissons" introduced by Professor Budin in Paris.

The little book before us contains four chapters, or lectures of instruction to the staff employed, and it is needless to say are replete with valuable information for all mothers, nurses, and district visitors. We earnestly hope that the system inaugurated at St. Marylebone will be generally adopted by all borough and urban councils. Dr. Pritchard is to be congratulated on the very efficient and comprehensive manner in which he has taught the important lessons which it is his duty to enforce, and it would be delightful to know that English mothers were ready and willing to learn them. That they fail to learn them is largely due to indolence and ignorance, but that they are capable of learning them, or at least some elementary principles of them, is evidenced by the fact that, in one of our large towns in the north, the mortality of infants under one year of age has been largely checked by the influence of a money prize to those mothers who have preserved their infants alive for 12 months. The question, however, is one of national importance, and the public spirit, evinced by the borough of St. Marylebone and its medical officers, is deserving of all praise.

Health Talks about Children. An Appeal for the Child. By JOHN GRIMSHAW, M.D., B.S. (Lond.), D.P.H. (Camb.), M.R.C.S. (Eng.), etc. Pp. 83. Birkenhead: Brimmell's Printing Works. 1s. net.

IN view of a declining birth-rate, and the excessive mortality among young children, too much cannot be said in favour of such valuable little books as Dr. Grimshaw's *Health Talks*. These Talks deal with the proper feeding, natural and artificial, of babies, with the ailments to which they are specially liable, with the need for social reform in reference to children, and are, of course, designed for the information of those who have the care of infants and young children. They will also be found eminently useful by those whose duty it is to give lessons on the hygiene of the home. For such persons, this little book will be found to be an admirable little inexpensive text-book, for it contains a mass of useful information and advice. Dr. Grimshaw very rightly lays great stress on the need for absolute cleanliness in dealing with the food and clothing of very young children. The conditions of life, in large towns especially, render the securing of this a difficult matter, but if some of the salient points, to which Dr. Grimshaw refers, could be epitomised and published in a small pamphlet at the cost of a few pence and widely circulated by local authorities, much good might be effected. The book itself should be in the hand of the members of all county councils.

Preservatives in Food and Food Examination. By JOHN C. THRESH, M.D. (Vic.), D.Sc. (Lond.), F.I.C., Lecturer on Public Health, London Hospital, Medical Officer of Health to the Essex County Council; and ARTHUR E. PORTER, M.D., M.A. (Cantab.), Assistant Medical Officer of Health to Leeds. Pp. xv and 484. Plates 8. London: J. & A. Churchill. 14s.

DR. THRESH and Dr. Porter are to be congratulated on their impartial treatment of an important and difficult problem. It is true that, in their preface, the authors appear to consider that the dangers attending the use of preservatives have been "greatly exaggerated," but, on referring to the text, the reader will find that the arguments *pro* and *con* have been fully and fairly stated.

To the analyst, this book will be invaluable. All the latest methods for the detection and estimation of the chemical preservatives, commonly found in food, are given in detail, and the authors have recommended only those processes which they have themselves tested, and found to be workable.

The long list of legal cases and decisions at the end of the volume should prove of assistance to those concerned in the administration of the Food and Drugs Acts.

A Study in Nursing. By Miss A. L. BRINGLE, formerly Matron of St. Thomas's Hospital, Superintendent of the Nightingale Training School for Nurses, Superintendent of the Nurses at Edinburgh Royal Infirmary. London: Macmillan & Co., Limited. 1s. net.

WE have a most useful book, and one widely differing from any other yet written. Given to us by one who knows, and who has been over every step of the road, it is valuable alike to the probationer, sister and matron. The author says that "there is nothing difficult in nursing, but the whole requires careful consideration," and she has herself evidently bestowed much attention to the subject, and, consequently gives a great deal of excellent advice to matrons and ward sisters. She has also much to say to those wishing to become nurses, and does not minimise the trials of the new probationer. In the first chapter, "What is Nursing?" the qualifications—mental and physical—required by candidates for this high profession are dwelt upon. For instance, the "least reflection shows that there must be labour and drudgery in the task of nursing; that it involves some offices that are strongly disagreeable" before anyone can "acquire knowledge" and "maintain attention" so necessary in all illness. The profession, however, is interesting and elevating, and "not a work to be relegated to those who are fit for nothing else." Throughout the book there is a high tone, so desirable in an age when utilitarianism is apt to dim the nobility of the grandest profession women can adopt. It is a book every nurse will find most helpful.

The Medical Diseases of Egypt. By F. M. SANDWITH, M.D., F.R.C.P., Consulting Physician to H.H. the Khedive, and to Kasr-el-Ainy Hospital, Cairo; Lecturer at the London School of Tropical Medicine, etc.; formerly Vice-Director of the Sanitary Department of Egypt. Part I. Pp. 316. London: Henry Kimpton.

IN this, the first part of Dr. Sandwith's treatise on the medical diseases of Egypt, the infective and parasitic diseases are dealt with. A uniform plan of treatment has been adopted for each disease; first, the synonyms and Arabic equivalents of the name are given; then a short history of the disease, its distribution, causes, contagion and varieties, symptoms and course, with clinical histories of a few typical cases, and often a temperature chart, the morbid anatomy, diagnosis and treatment, and, lastly, references to the literature. The book is a very valuable one for students of tropical medicine, and can also be recommended to practitioners at home. Not many British physicians, for instance, could give an account, first hand, of typhus and relapsing fevers. The chapters on bilharziosis, ankylostomiasis, and pellagra are particularly good.

Consumption: Its relation to Man and his Civilisation; its Prevention and Cure. By J. BESSNER HUBER, A.M., M.D., Fellow of the New York Academy of Medicine, Member of the National Association for the Study and Prevention of Tuberculosis, etc. Pp. 536. 131 illustrations. London: J. B. Lippincott Company. 15s. net.

IT is not an easy matter, in a short review, to convey an adequate idea of the volume before us. The book, as its title denotes, is a comprehensive exposition of the effect which consumption has had upon civilisation, and a consideration of its relation to human affairs. It will, therefore, be at once seen that its scope is much wider than that of an ordinary medical treatise; but, at the same time, it is an admirable *résumé* of all the latest developments of the study of tuberculosis, and of the measures, which are recommended, for its prevention and cure. It, of course, deals primarily with the conditions, which prevail in New York and other large American cities, but as these conditions are essentially similar to those, which exist in the crowded populations of other countries, the consideration of them is of paramount interest to all members of the profession and to social reformers.

The work is divided into 15 parts, each part being devoted to the special consideration of a particular branch of the subject, and, in its turn, being subdivided into from 3 to 8 very readable short chapters. Not the least interesting of these parts are those devoted to the accounts of the establishment and management of sanatoriums in America and the various countries of Europe. The volume is embellished with 131 exceedingly interesting and helpful illustrations, and is remarkably well printed on excellent paper, but we could wish that the weight of it were less ponderous, for few readers would like to study it for long unless it rested on a reading easel. For matter and style we can confidently recommend it to all interested in the subject.

Clinical Applied Anatomy, or the Anatomy of Medicine and Surgery. By CHARLES R. BOX, M.D., B.S., B.Sc. Lond., M.R.C.P. Lond., F.R.C.S., Eng., Physician to Out-patients St. Thomas's Hospital; Lecturer on Applied Anatomy, etc., St. Thomas's Hospital; and W. McAdam Eccles, M.S. (Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Joint Lecturer on Anatomy, and Demonstrator of Operative Surgery, St. Bartholomew's Hospital, etc. Pp. 471. London: J. & A. Churchill. 12s. 6d. net.

THIS book has been written more or less upon the lines suggested by the classical work *Rest and Pain*, by John Hilton, which appeared so long ago as 1862. The authors have throughout endeavoured to insist upon the importance of a sound anatomical knowledge to the practitioner, and they have been successful in emphasising the relationship of anatomy to both medicine and surgery. The book has been written, chiefly from the clinical point of view, and we can with confidence recommend it to both the general practitioner and senior medical student as a thoroughly trustworthy text-book, the need of which has long been felt. The book is illustrated by forty-five plates, some of which are coloured, and all of which are admirably produced.

Nervous and Mental Diseases. By J. D. NAGEL, M.D., Consulting Physician to the French Hospital of New York, &c. London: Hodder & Stoughton.

WITHIN the narrow limits assigned to him, the author of this volume has succeeded in presenting a clear exposition of his subject. As in most condensed accounts, the commoner diseases receive relatively less attention than the rarer. The first part, dealing with nervous diseases, needs an introduction to establish general principles and to co-ordinate what follows. The arrangement is, consequently, faulty. For instance, at the beginning of the book, under the heading of Diseases of the peripheral nerves, an account is given of anæsthesia, while towards the end muscular spasm is described under Cerebral Disorders.

The second part, that on Mental Diseases, has an introductory chapter, and is altogether better than the former. It concludes with a good appendix on Insomnia.

Hints on the Management of the Commoner Infections. By R. W. MARSDEN, M.D., M.R.C.P., D.P.H., Honorary Physician to the Ancoats Hospital, Manchester, etc. London: Wm. Heinemann. 3s. 6d. net.

WHEN called upon to deal with ordinary infectious diseases, practitioners, especially those in isolated country districts, are glad to have their opinions as to methods and treatment confirmed and fortified. To such, and to students, Dr. Marsden's little book will be exceedingly welcome. It is divided into three parts, (1) General Measures; (2) Specific Infections, of which 27 are ably dealt with; and (3) Appendices (Isolation and Disinfection). It is a book that few men in general practice can afford to be without.

The Care and Management of Delicate Children. By Dr. PERCY LEWIS, Honorary Medical Officer to the Victoria Hospital and Surgeon to St. Andrew's Convalescent Home, Folkestone. Pp. 102. London: Cassell & Co., Limited.

THIS little book is stated to be specially designed for "medical officers of schools and others to whom the care of delicate children is committed," and to these it may possibly be of some service, for it contains some useful hints and directions, and includes a system of medical gymnastics as an appendix. It does not contain much that is original, but the information is sound and trustworthy.

Notes on General Practice. By S. M. HEBBLETHWAITE, M.D. Lond., &c. Pp. 74. London: The Scientific Press, Limited.

IN spite of the three reasons which the author gives in his preface for publishing these notes, we are still left wondering why this little work was printed. No less than five pages are devoted to a description of urine testing! The remaining sixty-nine contain a heterogeneous collection of scraps, roughly sorted into heaps. There is always, however, a certain amount of interest in knowing what other men are doing, hence the hour or so required for reading through this booklet cannot be said to have been ill-spent.

A System of Surgical Nursing: with an Appendix containing useful Formulæ, Emergency Drill, &c. By A. N. McGREGOR, M.D., F.F.P.S.G., Assistant Surgeon to the Glasgow Royal Infirmary Pp. 554. Glasgow: David Bryce & Co.

A PORTLY volume, whose appearance is apt to strike one with alarm, seeing that it deals solely with surgical nursing. The information is very full and complete, and a surgical nurse, who has read and inwardly digested this system, will certainly prove, *cæteris paribus*, a most valuable assistant to an operator. We congratulate the author on the fact that the idea of nursing, *qua* nursing, is paramount throughout, and he does not afford the least excuse for any attempt at unqualified practice on the part of the nurse, while insisting on the necessity for an intelligent appreciation of the fundamental principles involved.

A Text-book of Physiology for Medical Students and Physicians. By WILLIAM H. HOWELL, Ph.D., M.D., LL.D., Professor of Physiology in the Johns Hopkins University, Baltimore. Pp. 905. Illustrated. London: W. B. Saunders & Co.

IN this work, the author has stated, as simply and lucidly as possible, the more important facts of physiology, has not discussed at length all the various theories upon any particular part of the subject, but has carefully chosen for consideration those which, in his opinion, possess especial interest. There are few illustrations, but those which are included are good. Throughout the book, there are numerous references to the literature of the subject, which cannot fail to be exceedingly useful to earnest and painstaking students, as well as to those who desire to add to their stock of knowledge.

Lectures on Midwifery for Midwives. By A. B. Calder, M.B., M.R.C.S., Lecturer on Midwifery to the London County Council, etc. London: Baillière, Tindall & Cox.

We have in this book a new and comprehensive work for midwives. The student and midwife will find it a handy book of reference as it is a recapitulation of lectures given "just in the words they were delivered to the class." The anatomy of the pelvic organs is dealt with in the first chapter. The four following chapters give an account of the symptoms and progress of pregnancy the mechanism and phenomena of labour. Three chapters are given to the management of labour, and are pre-eminently practical. The advantage of breast feeding over artificial feeding for infants is strongly advocated, but, as artificial feeding is sometimes inevitable, an outline of the infant's dietary is given in Chapter X. After dealing with sepsis and asepsis in Chapter XI., three chapters are given describing the symptoms and abnormalities, while sanitation is discussed and the Midwives Act explained in the concluding chapters. The illustrations cannot be so highly commended. Were it not that they are indexed and explained, even the initiated might be much puzzled. They are much too crowded.

A Handbook for Midwives and Maternity Nurses. By Comyns Berkeley, B.A., M.B., B.C. (Cantab.), M.R.C.P. (Lond.), M.R.C.S. (Eng.), Assistant Obstetric Physician to the Middlesex Hospital, etc. With 58 illustrations. London: Cassell & Co., Ltd.

A very excellent little book intended for those studying for the midwives' examination, but one that all qualified midwives would do well to possess. It is most complete, and so excellently arranged that the nurse or midwife can, without difficulty, find the cause, signs, symptoms, treatment, or result of any detail she may wish to verify. The division of the chapters under two headings, (1) Physiology, (2) Pathology, makes it particularly easy as a book of reference. The first part, consisting of four chapters, is devoted to anatomy. Part II. has six chapters on the physiology and pathology of pregnancy. Labour is treated in the same way in Part III., while the puerperium and the infant occupy Parts IV. and V. House sanitation, asepsis, and antisepsis make a fitting conclusion to a book which cannot be too strongly recommended to nurses. The illustrations are excellent and numerous.

The Treatment of Gonorrhœa in the Male. By Charles Leedham-Green, M.B., F.R.C.S., Senior Surgeon to Out-patients, Queen's Hospital, Birmingham, etc. Pp. xii + 151. 36 illustrations. London: Baillière, Tindall & Cox. 5s. net.

The treatment of gonorrhœa is a never-failing source of discussion, there seems to be no end to the number of methods which may be adopted, or of the number of drugs which have been used, each being advocated, at least for a time, as the best. In the book before us, the author gives a clear and concise account of the methods he has used, and which he has found to be useful. He rightly insists on the importance of finding out what part of the urethra is affected, and the methods by which such knowledge can be obtained. A practical account of the treatment to be adopted is then fully given according to the stages of the disease. A section is devoted to the complications, and there is a chapter on the proof of the cure of gonorrhœa, and its bearing on marriage. Throughout, the book will be found to be a very useful guide in the treatment of this troublesome complaint.

Elementary Manual of Regional and Topographical Dermatology. By R. SABOURAUD, Director of the City of Paris Dermatological Laboratory, St. Louis Hospital. English Translation by C. F. Marshall, late Assistant-Surgeon to the Hospital for Diseases of the Skin, Blackfriars, London. London: Rebman, Limited. Pp. 660. 21s.

THE author has attempted, in this work, to present the student with the problems of dermatology from an entirely new point of view. The intention is to enable a student, who knows nothing at all, to arrive at the correct diagnosis of a skin disease, by merely seeking his information under the heading of the part affected. It is a little doubtful whether, in the absence of all previous knowledge, this is a possibility, since most teachers have found that the difficulty lies, not in the mere academical knowledge, but in the making of accurate observations of the condition of the skin. Nevertheless, if a certain capability for examining the skin has already been acquired, this book might be extremely useful. The descriptions in all cases are good examples of Sabouraud's clear and forcible exposition, and though one may sometimes differ from him, it is impossible to misunderstand him. The translation is here and there slightly awkward, as if the translator, in his endeavour to retain Sabouraud's style, had adhered a little too closely to the French idiom. On the whole, however, it is well done, and certainly bears the true ring of Sabouraud's note. Printer's errors are rather numerous, which will no doubt disappear in a second edition. The book is well and copiously illustrated with reproductions of photographs.

Public Health Administration in Glasgow. A Memorial Volume of the writings of James Burn Russell, B.A., M.D., LL.D., Member of the Local Government Board for Scotland, and formerly Medical Officer of Health for Glasgow. Edited by A. K. CHALMERS, M.D., Medical Officer of Health for Glasgow. Glasgow: James Maclehose and Sons. 7s. 6d. net.

ALTHOUGH this volume deals largely with the history of the efforts made for the improvement of the sanitary condition of the City of Glasgow, during a period of more than half a century, it may also be regarded as a text book of Sanitary Science, and of all social questions closely allied to it. Dr. Russell seems to have been singularly well qualified for the important duties which he discharged with such advantage to the City, which had the good fortune to enjoy the benefits of his guidance and advice. By the publication of this memorial volume, all who are interested in the question of sanitation may participate in the enjoyment of these benefits. Chapters on the Prevention of Tuberculosis, on Disinfection, and on Meat Infection add to the value of the book, and a very beautiful portrait of Dr. Russell, in photogravure, forms a fitting introduction to it.

Dr. Chalmers is to be congratulated on the ability with which he has performed his duties as Editor.

NOTES FROM FOREIGN JOURNALS

ANTITHYROIDIN.

Elsner and Wiseman make, in the *New York State Journal of Medicine*, a report on the therapeutic value of antithyroidin in the treatment of Graves' disease. Their conclusions may thus be summed up:—

1. There was high tension of the pulse in all their cases, both before treatment, when the pulse was small and thready, and after treatment, when its tone and character had improved.

2. It must be borne in mind, when considering the efficacy of antithyroidin, that, of late years, a more favourable prognosis has been given than formerly in the disease, that there is a marked tendency to remission of symptoms lasting for some time (years in some cases); and that acute exacerbations of symptoms are not infrequent.

3. The remedy can certainly be used for the relief of the more alarming symptoms in all cases. The symptoms most relieved are, tachycardia, praecordial distress, and tremor. Rest in bed and careful dieting hasten the improvement, but, without antithyroidin, do not yield such an amount of cardiac comfort.

4. Improvement in one or more of the symptoms usually follows within from three to seven days after beginning the treatment. If no improvement has been gained after three to four weeks, it is extremely unlikely that any ultimate benefit will be obtained. In serious cases, the treatment must be continued for several months. In all cases, when the subjective symptoms have disappeared, it is wise to give antithyroidin for periods of from four to eight weeks at intervals of two or three months.

5. The cases in which the best results have been obtained have been without marked goitre and with slight exophthalmos, tremor, and Graefe's symptom. The enlarged thyroid has decreased in size, but has not returned to the normal.

6. The most rebellious symptom is exophthalmos, which has never yielded entirely to antithyroidin treatment.

7. Nervous symptoms subside, as a rule, when the heart's action gets slower, and the patients are relieved, at the same time, of their many fears.

8. Most of the patients gained in weight. Those who have taken the remedy for a long period feel the loss of it greatly when it is discontinued, and are very glad to return to its use.

9. The remedy has always proved itself harmless, and may be taken in pregnancy. Hypertrophied and dilated hearts do not contra-indicate its use.

The serum was given in doses of 15 to 30 minims, night and morning, and, if no benefit resulted from the small dose, it was increased to 40 or even 60 minims. It is taken by the mouth in water, syrups, or light wine. The number of cases observed is too small to admit of definite statements being made, but the authors are encouraged by their experience to give the serum a thorough trial, believing that the treatment will ultimately prove to be a rational therapeutic method.

ACCUMULATIONS OF WAX IN THE EAR.

Lermoyez points out, in the *Gazette Médicale de Paris*, that, when the wax is soft, the ear should be syringed out with a warm solution of bicarbonate of soda (about gr. v. in ℥j). The injection must be directed along the postero-superior wall of the external canal. In the case of a hard mass of wax, 10 to 15 drops of warm oxygenated water should be instilled, and left to act for half an hour at least before syringing. Failing this, an ear-bath, lasting 10 minutes, should be given three times a day with—

℞. Sodii Bicarb.	gr. x.
Glycerini	
Aq. destill.	āā ℥ss.

Between the baths, the meatus must be closed with a plug of non-absorbent wool. After 48 hours, syringing will be successful.

METHYLENE-BLUE IN CANCER.

In the *Journal of the American Medical Association* for November, Jacobi gives his experience of the use of this drug (methyl-thionine hydrochloride) in the treatment of cancer, especially in inoperable cases. He has been using it internally for fourteen or fifteen years, as he found injections too painful, and he follows up all his operative cases with this treatment. He does not claim any cures, but has seen patients keep comparatively well for years under this treatment. He is now combining it with light-treatment, but cannot as yet give any positive results. He thinks, however, that the combination will prove to be more effective than methylene-blue alone. He gives the drug in pills, beginning with a dose of 2 grains a day, and gradually increasing to 6 grains. The amount used should be made up with three-quarters of a grain of extract of belladonna, to counteract the disagreeable dysuria, and divided into three or four doses. He frequently combines with it arsenious acid, in doses of $\frac{1}{20}$th to $\frac{1}{10}$th of a grain three times a day, preferring this preparation to Fowler's solution. Strychnine may also be combined. The patient must be warned that the urine will be blue, and will stain linen indelibly. Intra-abdominal cancers do very well under this treatment. Jacobi has had one case of cancer of the liver under observation for eight years, and the patient is still fully able to attend to his business.

A MOUTH-WASH.

In the *Bulletin général de thérapeutique*, the following is recommended by Le Gendre as useful:—

℞. Thymoli	gr. ss.
Acid. Benzoic.	gr. xv.
Tinct. Eucalypti.	ʒi.
Aq. destill.	ad ℥xij.

M. Fiat lotio.

THE NUTRITIVE REQUIREMENTS OF THE BODY.

The *Therapeutic Gazette* for December contains a leading article upon this subject, in which attention is drawn to the communication made by Benedict to the *American Journal of Physiology* in August. Up to the time of Chittenden's experiments, it was an accepted fact that the human body requires a quantity of food sufficient to provide from 3,000 to 3,500 calories daily. Chittenden has given several lectures on the subject during the past year, and has shown the state of nutrition of athletes, soldiers, professional men, and others, who have followed the low diet he advocates. Benedict points out that, as a rule, human beings devour the quantity of food which they desire, and the varieties which are agreeable to them. In estimating the quantity of food necessary, it must be remembered that there is some difference between the quantity taken in and the quantity actually digested and absorbed. Food, of course, should be ingested in that amount which is sufficient to repair the waste of the body, to furnish the necessary amount of energy for purposes of work and warmth, to keep the body in full vigour, and, in the case of the young, to provide the excess proper for normal growth.

Recent investigations seem to prove that the quantity of previously formed protein tissue, which is broken down each day, is relatively small. But in addition to tissue protein, there is in the body a large supply of fluid protein not actually incorporated into the material of the living cell, and very large amounts of this fluid proteid may be used up daily in the food values. The average amount of protein, taken by men following sedentary occupations, is about 100 grammes a day, and the demand for protein increases as muscular work increases. Benedict asserts that the upper classes in Japan get liberal amounts of protein, and these are they, who have done most towards placing the country in the present great position.

Benedict quotes experiments which show, that when hogs and cattle are deprived of the normal quantity of protein to which they are accustomed, they seem to remain in perfect health for some months, but ultimately their health is seriously impaired, and they do not recover until generous amounts of protein are restored. As illustrating the deleterious effects of small quantities of protein, he cites the case of the poor white, and the poor negro of the South, and the Italian labourer of Southern Italy.

Another interesting confirmation of the belief, that considerable quantities of protein are necessary for health, is found in the experiments carried out by the Medical Department of the Japanese Navy. One battleship made a long voyage, with the crew on a ration containing only 91 grammes of protein a day. On the return of the ship, the health of the crew was materially impaired, and beri-beri had been frequent. Another battleship made exactly the same voyage, with the crew receiving twice as much protein. The crew returned in first-rate physical condition, and there had been very little beri-beri.

Benedict carried out some calorimetrical experiments upon a person using comparatively small quantities of food. The heart output, at rest, was found to be about 1,800 calories a day, while moving about a little brought the output up to over 2,000 calories a day. The value of the food taken by the subject only amounted to 1,400 calories, so that the difference had to be provided from the patient's body. This loss was not shown on weighing, but Benedict points out that the loss of a few pounds of fat may be readily compensated by the gain of a few pounds of water.

The general conclusion arrived at is that Chittenden's experiments are of interest, as showing what can be done, rather than what ought to be done. Permanent reductions in the total food intake are decidedly disadvantageous, and not without possible danger. The energy intake cannot be diminished without diminishing the energy output. Large amounts of energy are necessary for expenditure in the action of the heart, the respiratory muscles, and other vital processes. We therefore return to our former belief that the food required must be sufficient to provide an individual with about 3,000 calories a day, so as to keep him in perfect health.

INHALATION FOR CHRONIC BRONCHITIS.

The following is recommended, in the *Bulletin générale de thérapeutique*, for use in an inhaler, so arranged that the air must pass through the mixture, and thus become saturated :—

℞. Ol. Eucalypti - - - - - ℥iss.
Thymol. - - - - - ʒi.
Menthol.
Guaiacol. Crystall. - - - āā ʒii.
Aq. - - - - - ℥x.
M.

TREATMENT OF RHINO-PHARYNGITIS IN CHILDREN.

Le Marc Hardour, in the *Revue internationale de médecine et de chirurgie*, states that he considers that the best way to treat rhino-pharyngitis in children is to introduce into the nostrils, three or four times a day, tampons of absorbent wool drawn out to a point and covered with boric vaseline. The child at first sneezes, then puts up with the presence of the wool, and sniffs in, whereby some of the vaseline penetrates into the nasal fossæ as far as the pharynx.

Astringents like alum, tannin, and especially antipyrine, may be added to the boric vaseline :—

℞. Acidi Borici - - - - - gr. xx.
Phenazoni - - - - - gr. x – xx.
Vaselini - - - - - ℥j.
Misce. Fiat unguentum.

For children who cannot tolerate the tampons, menthol in oil may be used ; one or two drops of the following being introduced into each nostril night and morning :—

℞. Menthol - - - - - gr. vijss.
Ol. Amygd. dulc. - - - - ʒj.

Older children may use one of the following powders :—

℞. Aristol,
Lactosi - - - - - - - āā ʒj.
Or,
℞. Aluminii Aceto-tart. - - - ʒj.
Lactosi - - - - - ʒiss.

The mouth and nose should be sprayed (steam-spray) with boric-acid lotion, normal salt-solution, Enghien water, Challes water, or Mont-Dore water.

If these measures do not suffice, the walls of the pharynx must be directly acted upon by painting with glycerine and iodine, or with the following :—

℞. Iodi. - - - - - gr. v.
Potass. Iodid. - - - - gr. xl.
Ess. Menth. Pip. - - - - ♏v.
Glycerini - - - - - ℥j.

This should be used twice a week at first, afterwards once, upon wool carried on a curved probe, which can be passed up behind the soft palate.

As the majority of children suffering from this affection are "lymphatic," general treatment must be adopted by means of cod-liver oil, iodo-tannic syrup, and good feeding.

TREATMENT OF THE SYPHILITIC CORYZA OF NEW-BORN INFANTS.

Coryza is often the first symptom of infantile syphilis, and sometimes even remains as the only one for several weeks. In an article in *La Clinique*, Georges Laurens points out the necessity for early diagnosis and prompt treatment so as to prevent the extension of the lesions, and the destruction of the nose, leading to the typical deformity. Treatment must be local and general, for each is of importance. As regards local measures, the first thing to do is to "aspirate" the nasal cavities by means of an india-rubber ball with an appropriate nozzle just before the child is put to the breast for each feed. It is the application of the "vacuum cleanser" to the rhinitis of the new-born, but can only be done if the mucus is sufficiently fluid. Oxygenated water, plain, or diluted with an equal quantity of boiling water, must be instilled into the nostrils several times a day. This is best managed by pouring the small amount, four or five drops, into the nostril out of a small teaspoon. Reflex sneezing is produced and the nose is cleared by the expulsion of "candles." The toilet of the nostril is completed by the use of sterilised oil of vaseline, a few drops of which are instilled into the nostrils several times a day so as to lubricate the mucous surfaces.

Adrenalin is very useful for re-establishing the permeability of the air passages.

℞ Liq. Adrenalin. Hydrochlor. 1 p$^{0}/_{00}$ - - ♏xx.
Aq. destill. et steril. - - - - - ℥j.

Three or four drops of this solution should be introduced into the nostrils about five or ten minutes before each feeding so as to facilitate respiration during the meal. If these measures are carefully carried out, the nasal fossæ will be rendered relatively aseptic, and secondary infections will be prevented.

As regards general treatment, the mother will be placed under specific treatment, and the child will undergo inunction, using for this one gramme of mercury ointment. The skin of the flanks and chest should be well washed with soap and the ointment then gently rubbed in. It is left on all night covered with a layer of wool and washed off in the morning. Daily applications should be made until cure is complete, paying due regard to the gastro-intestinal tract.

FOR URTICARIA.

Gaucher recommends the application of the following to allay the intense itching of urticaria :—

℞. Mentholi - - - - - - ʒiiss.
Chloroformi,
Aetheris,
Spirit. Camphoræ - - - - - - - āā ℥j.
M. For outward application.

MENTHOL IN GASTRIC LESIONS.

Amblard, for three years under Huchard, has been investigating the action of menthol in gastritis. The drug produces an anæsthetic effect upon the gastric mucous membrane, and its use to allay pain in the stomach is easy, and not harmful, being preferable to cocaine, the use of which should be given up on account of its toxicity. The effects produced by menthol may be explained in three ways:—

1. Its anæsthetic action allays pain and vomiting.
2. Its antiseptic property influences the gastric milieu.

3. It produces in the mucous membrane a congestion of such a nature as to alter painful symptoms and vomiting.

Amblard recommends its use in dyspeptic conditions, which do not appear to have any connection with an organic lesion of the stomach, but are due to disorders of the nerve supply and of the gastric secretion. Good results are obtained in the dyspepsia of chlorosis, and of pulmonary tuberculosis in the gastric symptoms of neurasthenia, the gastric crises of tabes, and the obstinate vomiting of pregnancy. In gastric affections, ulcer and cancer, the use of menthol yields good results, and does not induce hæmorrhage. Leaving off the drug is followed by a return of the pain and vomiting.

Amblard uses fractional doses, giving gr. iss. to iij. in a day. The susceptibility of the patient must be tested, and it is well to begin with the following:—

℞. Mentholi - - - - - gr. $\frac{1}{3}$
Sacchas. Lactis - - - - gr. viiss.
M. In cachet j.

Half a tumblerful of water should be taken after this has been swallowed. If the desired result is not obtained, the dose must be increased to $\frac{5}{8}$th of a grain. (*Journal des Praticiens.*)

TREATMENT OF PNEUMONIA.

Robin considers that a really rational treatment of pneumonia is yet to find, and has sought for information among the phenomena observed at the period of defervescence. At that time there is a considerable excretion of urea and uric acid, and if this is noticed to occur before the temperature falls, it is a favourable omen. The hydrating and oxydro-reducing diastases, serums, and metallic ferments promote the natural tendency of the organism to cure itself, and therefore help should be looked for among these. Of these, the metallic ferments offer the greatest advantages, for the use of these by injection increases the elimination of urea and of uric acid, the maximum effect being obtained 24 to 72 hours after injection. This action upon the pneumonic lesion itself is nothing, so that they can only be an element in treatment to which others can be added in case of need; as, for example, bleeding in some cases, calomel in fractional doses, moderate amounts of alcohol, small doses of bi-hydrochloride of quinine combined with pyramidon, beginning on the fourth day of the disease, and, finally, blisters from the fifth day.

These ferments bring about, almost invariably, a decrease in the temperature, a special urinary reaction, and a slight increase in arterial tension.

Out of 53 cases treated on the above lines, 26 of which were of great severity, the mortality has only been 6 cases, or a percentage of 11·32. In 63 per cent. of these cases, the crisis happened before the eighth day.

In the case of broncho-pneumonia, secondary and sinous, the treatment was less active, for out of 13 cases, all very severe attacks, 6 deaths occurred, a percentage of 46.

The ferments were given, by deep hypodermic injections, in doses of 10 c.c., or, as in the most serious cases, by intravenous injections of 5 c.c. The nature of the metal appeared to be a matter of indifference. The injections should be begun on the fourth day of the disease, and continued every other day.

FOR PRURITUS.

Brocq recommends the use of the ointment "*aux trois acides*" :—

℞.	Acid. Carbolic	♏xv.
	Acid. Salicylic	gr. xxx.
	Acid. Tartaric	gr. xlv.
	Glycerin. Amyli	℥ij.

M. Ft. ung.

TO REPLACE THE BELLOWS OF THE THERMO-CAUTERY.

The *Gazette Médicale de Paris*, in calling attention to the fact that the india-rubber bellows of a "thermo-cautère" perishes very quickly, and that this is often very embarrassing for a practitioner, points out that a very simple procedure is to replace the bellows by a bicycle-pump. Some experiments have just been made, which prove this to be a most efficient substitute.

TREATMENT OF HAY-FEVER.

The *Journal de Médecine et de Chirurgie pratiques* extracts the following. First among the methods, for relief of the attacks of hay-fever, are placed inhalations of menthol in chloroform (4 per cent.); next to these, are intra nasal sprayings with 1 in 2,000 solution of adrenaline. Mydriatics should be used for the eye-symptoms, especially for photophobia.

℞.	Eserin. Sulph.	gr. j.
	Aq. destill.	℥j. M.

"Instil one drop of this solution into each eye;" or

℞.	Pilocarpin. Nit.	gr. iiss.
	Aq. destill.	ʒj. M.

"To be used in the same way."

In asthmatic forms, the dyspnœa must be treated with antispasmodics. In slight cases, the use of belladonna or valerian is indicated :—

℞.	Ext. Belladonnæ	gr. ¾.
	Syrup. Rhœados	℥j.
	Aq. Laurocerasi	℥ss.
	Aq. Tiliæ.	ad ℥iv. M.

To be taken in the course of the day, divided into four doses; or

℞.	Zinci (vel Ammonii) Valerianat.	
	Ext Valerian.	āā gr. ¼.

M. Ft. pil j. Mitte tales nº. xx. Cap. j 6tis vel 4tis horis.

When the asthmatic attacks are more severe, recourse must be made to the bromides, giving the following :—

℞.	Potassii Bromidi	ʒv.
	Tinct. Lobeliæ.	℥ss.
	Tinct. Grindeliæ Robustæ	ʒj.
	Tinct. Camph. Co.	ʒiij.
	Syrup. Aurantii	℥ss.
	Inf. Senegæ.	ad ℥viij.

M. Ft. Mistura. Cap. ℥ ss. ter quotidie.

If, however, the attacks of asthma are very severe, injections of morphia or of heroin must be used. Search should always be made to see if any local lesion is present in the nose independently of the hay-fever.

Notes by the Way.

The Grand Old Man of Surgery.

THE medical profession throughout the world has combined to do honour to Lord Lister on the occasion of his completing his eightieth year, and THE PRACTITIONER joins fervently in the hope that he may live to celebrate many returns of a birthday so auspicious for mankind. It is pleasant to know that the Grand Old Man of Surgery is still in the fulness of his intellectual vigour, and is taking the keenest interest in the re-issue in permanent form of the writings which have revolutionised surgical science and expanded the art to an extent undreamed of by the most advanced surgeons only a generation ago. His doctrines did not at once secure acceptance. The men who had reached what Osler calls the "fixed period" treated the complicated ritual of antisepsis as foolishness, and spoke, more or less openly, of the theory on which it was founded as moonshine. The modifications of his original methods, necessarily brought about by larger knowledge and experience, were ignorantly cited as proofs of the unsoundness of the foundation. It is a suggestive coincidence that a similar line of argument is used against vaccination. Lister, however, was more fortunate than many of the men who have made far-reaching discoveries, for he was neither ignored nor persecuted. He has lived to see himself universally revered as the *Maestro di color che sanno*, and his name among the very highest of those who have stolen the fire of new truth from heaven to benefit their kind. Doubtless he would himself be content with the veneration of those who can appraise the value of his work. But it is gratifying to his brethren that he has not been without the honour which the prophet seldom finds in his own country.

* * * * *

Medical Fame.

FEW indeed, outside the fold of medicine, know the greatness of the man or understand what he has done for his fellow men. He has never sought the praise of the multitude, and his name does not cause the "great heart of the people" to throb as does that of a tenth-rate general, or a champion wrestler. He was one of the first on whom the Order of Merit was conferred, and his inclusion in the list of those so honoured seems to have perplexed the minds of some superior persons. Reference was made at the time in THE PRACTITIONER to the significant fact that, in certain alternative lists that were proposed in the papers, the name of Lister—the one as to which it might have been thought there could be no possible difference of opinion—was conspicuous by its absence. It must sorrowfully be confessed that Philistinism is as much the characteristic of the British people as before Matthew Arnold tried to infuse sweetness and light into his countrymen. A short time ago a Paris newspaper invited the French public to vote on the question, Who was the greatest Frenchman of the nineteenth century? Pasteur headed the poll by an enormous majority, leaving Napoleon, Victor Hugo, and other popular favourites far behind. One can imagine the result of such a vote in this country. Lister, beyond all comparison the greatest Englishman of his day, the man before whose work that of statesmen and generals pales into insignificance, would have been nowhere! Such is the fame that the prevention of suffering and the saving of life brings to the medical discoverer. We must comfort ourselves with the thought that, as Carlyle said to Lord Lister's great father-in-law, James Syme, whoever—archbishop, chancellor, prime minister—is in the wrong, the doctor is in the right. This applies to the humblest among us, as well as to the greatest.

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Death of Mr. Wakley.

THE news of the death of Mr. Thomas Henry Wakley, F.R.C.S., the late Senior Editor of *The Lancet,* who had just completed the eighty-sixth anniversary of his birthday, has been received with universal regret throughout the medical profession. Unfortunately, we have not the space at our command to give a biographical account of his professional life. We can, however, bear testimony to his uprightness of character, his kindliness of disposition, and his always earnest endeavours to forward the interests of the profession in every possible way. We may, with every justification, use the words of Marcus Antonius:

> "His life was gentle, and the elements
> So mix'd in him that Nature might stand up
> And say to all the world,
> 'This was a man.'"

* * * * *

The Use of Opium. IN the present issue the therapeutic uses and mode of administration of opium are discussed in a series of papers which, taken together, form, we think, a valuable addition to the existing body of accumulated knowledge on the subject. We would call special attention to Dr. Burney Yeo's contribution, as he speaks out of the fulness of an exceptionally ripe experience, and deals with the whole question in a broad spirit of what may be called clinical philosophy. This quality is by no means always conspicuous in medical writers. Professor Stockman deals with the subject in a manner highly helpful to the intelligent practitioner, as he sets forth with singular clearness and precision what an old French writer called the *pourquoi des choses*. It may be noted that he is disinclined to admit the validity of the common objection to the administration of opium in certain painful diseases, that it "masks the symptoms." As he points out, in perforation surgical intervention is necessary in any case, and it does not matter whether opium masks the symptoms or not. Again, in regard to acute peritonitis, he says well that the pain and discomfort complained of by the patient should not form the criteria of the necessity of an operation ; this should be decided by the local signs, the leucocytosis, temperature, pulse, and general condition. We are glad to have this expression of opinion from so high an authority, for we believe that, owing to an exaggerated fear of "masking symptoms," many patients are allowed to suffer much needless pain. Dr. Sandifer lays stress on the psychic action of opium and its power of modifying the maleficent influence of such mental states as fear, anxiety, and the depression caused by continued pain.

Dr. Bousfield shows how really severe pain may be discriminated from slighter degrees magnified by excitement, anxiety, or restlessness. Dr. Newman Neild describes a condition of much interest and importance which has hardly yet received adequate attention from clinicians. Professor Dixon's paper reveals the true inwardness of morphinomania, so that we can sympathise with the victim in his sufferings, while learning how he may best be delivered from his slavery. On the whole the papers, while not justifying De Quincey's biased tribute to "just, subtle, and all-conquering opium," confirm the traditional view that it is among God's best gifts to man.

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"Lancet" and Liquor.

The Lancet caused a sensation some weeks ago by publishing a manifesto on the use of alcoholic beverages, signed by a number of well-known members of the medical profession. The document set forth that the signatories were convinced of the correctness of the opinion that in disease alcohol is a rapid and trustworthy restorative. They added that in many cases it is truly life-preserving, owing to its power to sustain cardiac and nervous energy, while protecting the wasting nitrogenous tissues. They went on to express the opinion that "the universal belief of mankind, that the moderate use of alcoholic beverages is, for adults, usually beneficial, is amply justified." The one remarkable thing about the utterance was the time chosen for making it. If the manifesto was intended as a counterblast to certain words spoken in Canada last summer by an apostle of temperance, it was curiously belated. Yet, if it was not meant for this, what special reason was there which impelled the signatories to promulgate *urbi et orbi* a doctrine which is not in any way new, and is not put forward with any novelty of argument or illustration? It is, of course, out of the question to suppose that its appearance, simultaneously with a broadcast sending of samples of a certain brandy to medical practitioners, is anything more than an unlucky coincidence.

* * * * *

A Double Shuffle.

It must be owned, however, that if surmises of an unflattering nature were made about the origin of the document, those responsible for its production have only themselves to thank. It is amusing, though not particularly edifying, to note how some of those who signed it immediately hastened to explain away their action when they saw their declaration in cold print. In the face of the clear statement that "the universal belief of civilised mankind that the moderate use of alcoholic beverages is, for adults, usually beneficial, is amply justified," one of the signatories is reported in the *Daily Mail* to have made, to an interviewer, the following remarkable *apologia*:

"I hear that the declaration has been twisted to mean anything, justify anything; whereas the essential thing about it was to justify the use of alcohol in acute disease!" One must in charity suppose that he signed the document without reading it. At any rate, he will find it difficult to "twist" the words we have quoted out of their plain meaning. Some of the signatories stated that they would not have appended their names had they been aware that the manifesto was drawn up by a gentleman who is not of the household of medicine. This is surely a somewhat mean evasion. How much better is the attitude of those who stoutly stick to their guns!

* * * * *

The Triumph of the Invalid.

It has been well said that much of the best work of the world has been done by invalids. History is full of examples of the triumph of the spirit over a body racked with suffering. We doubt whether in the whole record of the human race any more striking illustration of this is to be found than in the case of Niels Ryberg Finsen. What he did is known of all men; but few know the conditions under which his work was carried out. These are set forth in a striking manner in a monograph entitled *La Maladie de Finsen*, which was recently presented to the Paris Academy of Medicine by Professor Antonin Poncet, of Lyons, on behalf of the author, Dr. René Leriche. The story is so full of interest, medical as well as human, that we make no excuse for summarising it here. Born at Thorshavn, in the Faroe Islands, on December 15, 1860, Finsen died at Copenhagen on December 15, 1904, having lived exactly forty-four years. Almost from his birth he was the prey, it might almost be said the sport, of disease. In his childhood he suffered from meningitis, pneumonia, acute nephritis, and other ailments. The long disease, which made the remainder of his existence a living death, began when he was 23. At first the symptoms were indefinite, and seemed to point to tuberculous peritonitis. For years his condition fluctuated between varying degrees of illness, exacerbations alternating with temporary improvement. His spleen and liver were enlarged, and this led him to believe that the cause of the mischief was the ecchinococcus, which is endemic in some of the places where he had lived. Two punctures of the liver, however, gave issue only to blood. The doctors who saw him thought of heart affection, following an old attack of articular rheumatism, and other things, but no definitive diagnosis seems to have been arrived at. About the age of 30, slight febrile phenomena were followed by the development of ascites, which attained such dimensions that in the later years of his life he was tapped eighteen times, and by pleural effusion. His condition became much worse. He suffered great pain, there was constant fever, and he lost flesh. He coughed incessantly, and at last died almost suddenly after a conversation with some of his friends and assistants.

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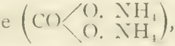
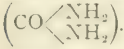
A Living Museum of Pathology.

DURING Finsen's life, his case puzzled all the medical authorities who were consulted, and the post-mortem examination, the record of which, by A. Flöystrupp and V. Scheel, may be found in *Der Therapie der Gegenwart*, for July 1905, did not clear up the mystery. There was general adhesion of serous membranes, and polyserotitis, with atrophy, about the spleen, and especially the liver. The heart and kidneys were sound. There were no tuberculous lesions in the lungs, but there were enlarged tracheo-bronchial and mesenteric glands. Dr. Leriche holds that recent researches on inflammatory tuberculosis probably furnish the explanation of the various pathological manifestations in Finsen's case. He thinks it certain that his disease was one of those forms of perivisceritis which of late years have been investigated by Bard, and particularly by Huchard. A close study of all the facts of the case, as recorded in a daily journal kept by Finsen himself, and of the results of the autopsy, conveys a very definite notion of an attenuated tuberculous infection, of the kind recently described by Leriche under the name of "tuberculous articular rheumatism" and "inflammatory tuberculosis." It would thus seem that by the irony of fate, Finsen, without any one suspecting it, fell a victim to the morbific agent over which he had so often been victorious in others. Whether or not we accept M. Leriche's explanation, it is none the less an amazing fact that one of the greatest advances in the healing art should have been by a man, who can scarcely have had a single day of healthy life, and whose body, for the greater part of its existence, was a living museum of pathology.

Practical Notes.

THE ORIGIN OF UREA:—It has long been recognised that urea is simply excreted, and not formed, by the kidneys, and that, for the most part, it is produced in the liver. What the precursors of urea in the body are has been a matter of considerable doubt, and, until quite recently *Creatine*, a substance found in muscle, and one of the substances, which represents the end-stage of the endogenous katabolism of protein material in the body, was looked upon as its chief precursor. More recently, however, it has been shown that the creatine, which is formed in the tissues of the body, and which represents the wear and tear of the nitrogenous tissues, is conveyed to the liver, where it is converted into *Creatinine*, and, as such, is eliminated in the urine, the amount of nitrogenous waste in muscle tissue which leaves it as urea is really insignificant. If creatine is introduced into the blood stream of an animal, the amount of urea excreted in the urine is in no way increased, but the output of creatinine is, showing that the precursor of creatinine in the urine is the creatine which is produced in the tissues. The antecedents of urea must, therefore, be sought for elsewhere. During protein digestion, as it occurs in the stomach and small intestine, the albumins and globulins, by the action of the pepsin-hydrochloric acid of the gastric juice, are converted into more soluble substances, such as the albumoses and peptones; and, in a similar way, by the action of the trypsin-sodium carbonate of the pancreatic juice, the albumins and globulins are converted into albumose and peptone. The peptone, thus formed, is, by the further action of the trypsin-sodium carbonate, and the erepsin found in the succus entericus, converted into certain cleavage products of protein digestion: these are ammonium compounds, leucine (α-amino-iso-butyl-acetic acid), tyrosine (oxy-phenyl-amino-propionic acid), aspartic acid, glutamic acid, and the bodies known as the hexone bases (lysine, arginine, histidine). These bodies form together short linkages of the amino-acids called the *poly-*

peptides. These substances are absorbed by the columnar epithelial cells, which are present on the free surface of the villi which practically line the whole of the small intestine. After these amino-compounds are absorbed into the blood-stream, they may be considered as the "building-stones" of which the more complex body tissue proteins are made.

Each tissue assimilates that particular amino-body which is most essential for its own use, and it has been surmised that tyrosine (oxy-phenyl-amino-propionic acid) and phenyl-alanine (phenyl-amino-propionic acid) are the most useful "building-stones" to the chief body proteins. The waste "building-stones," or amino-acids, those for which the body tissues have no use, are then converted into urea in the liver, and are hence excreted later. The urea thus produced is known as the *exogenous urea*. It has already been stated that most of the tissue creatine is converted into creatinine in the liver, but it is quite possible that a small quantity of the tissue creatine may be converted into urea and sarcosine (methyl glycine or methyl-amino-acetic acid). Sarcosine is then converted into ammonium carbonate  $\left(CO\begin{matrix} \diagup O.\ NH_4 \\ \diagdown O.\ NH_4 \end{matrix}\right)$, the ammonium carbonate is then further changed into ammonium carbamate $\left(CO\begin{matrix} \diagup O.\ NH_4 \\ \diagdown NH_2 \end{matrix}\right)$, and this, finally, in the liver into carbamide, or urea  $\left(CO\begin{matrix} \diagup NH_2 \\ \diagdown NH_2 \end{matrix}\right)$. The urea, which is formed in this way, is termed the *endogenous urea*. It has been shown, experimentally, that ammonium carbonate, ammonium carbamate, leucine, and glycine (amino-acetic acid), when injected into the portal vein of an animal, all cause an increased output of urea in the hepatic vein. This result does not occur when the same experiment is performed with any other organ in the body, hence these experiments prove the great importance of the liver in urea formation. Moreover, the blood in the carotid artery of an animal contains 2 milligrammes per cent. of ammonia, whereas the blood in the portal vein contains as much as 6 to 8 milligrammes per cent. of ammonia; this, of course, suggests that the liver is constantly changing ammonium compounds, and amino-compounds, too, into urea. If, in an animal, the portal vein is led into the inferior vena cava so that no portal blood gets to the liver, and the animal is then fed on a protein diet, the urine is found to contain much less urea than normal, but ammonium carbonate and ammonia are found instead. The animal later develops convulsions, passes into a state of coma, and dies of ammonæmia.

In persons suffering from acute yellow atrophy of the liver, it is found that the urine contains much less urea than is normal, its place being taken by a corresponding increase in the amount of ammonia present. Herter finds that it may be increased from the normal, from 2 to 5 per cent. up to as much as 17 per cent. The diminution of urea is probably partly due to the fact that the liver cells fail to manufacture urea from the ammonia supplied to them. Leucine and tyrosine are frequently present in the urine too. The present view, however, is that the leucine and tyrosine are derived from the liver-cells themselves as a result of their extensive destruction by the poison which produces the disease. It is said too that, in some forms of cirrhosis of the liver, there is a diminished output of urea in the urine, but there is an increase in the ammonium compounds present. From what has been stated, it will be readily understood that the estimation of the amount of urea excreted in a twenty-four hours specimen of urine can give us no true estimate of the amount of nitrogenous katabolism which is occurring in the body. The only way of getting any knowledge upon this subject is by accurately estimating the total output of nitrogen in the urine in twenty-four hours, and this, to be of any value, must be compared with the amount of nitrogen taken into the body as food. Kjeldahl's method is generally used for this purpose, for by it all the combined nitrogen in the urine is converted into ammonia, by means of sulphuric acid the ammonia can then be accurately estimated.

THE INFLUENCE OF OPIUM UPON THE EYE.—When a patient is under the influence of opium, the pupils are, as a rule, equal and very contracted. This is said to be due to the stimulation, by the drug, of the portion of the nucleus of the third cranial nerve, which supplies the sphincter iridis muscle. The action of the opium is, therefore, upon the central nervous system. The effect upon the pupil is much the same as that which arises during an extensive hæmorrhage into the pons varolii. If, however, a patient has taken a large dose of opium or morphia, and the case ends fatally, breathing becomes embarrassed, the pulse very irregular, and, just before death, the pupils become widely dilated. When a patient is suffering from morphinism, the pupils, except when the patient is under the direct influence of the drug, are widely dilated, occasionally they are unequal. In many respects, morphine and atropine may be considered as antagonistic drugs; in some of their actions this antagonism is real, as, for instance, their respective effects on the cerebral convolutions, respiratory centre in the medulla oblongata, and upon the intestinal movements. In fact this antagonism is apparent only, for the contraction of the pupil caused by opium and morphia occurs through their action upon the centre for the contraction of the pupil in the midbrain, whereas the dilatation, caused by atropine, is referable to the paralysis of the ciliary branches of the third cranial nerve at their periphery. Opium and morphia are diaphoretics, by virtue of their action upon the sweat centres, whereas atropine is an anti-idrotic, through its action upon the terminals of the nerves in the sweat-glands, that is, its action is a peripheral one. Both morphia and atropine, however, are cardiac depressants, and, in poisonous doses, cause a well-marked fall in arterial blood pressure.

SYMPATHETIC OPHTHALMITIS.—This condition is an acute serous or plastic inflammation of the uveal tract, which includes the iris, ciliary body, and choroid of one eye, due to a like inflammatory condition in the other eye, which is generally known as the *exciting eye*, whereas the eye affected with the sympathetic ophthalmitis is called the *sympathising eye*. The cause of the inflammation in the exciting eye is, as a rule, started by an injury which has resulted in a *perforating* wound. A foreign body impacted in the eyeball may be the cause. A wound through the corneo-scleral junction involving the iris and the ciliary body (the dangerous zone of the eye) is, perhaps, the most frequent cause of the disease. Sometimes it results from an iridocyclitis, which has been set up by the perforation of a corneal ulcer. Sympathetic ophthalmitis sometimes follows the operation for extraction of a cataract.

The condition may be met with at any age, but it is usually rather more frequently seen in young persons. The dangerous condition of wounded eye should be recognised early, and the eye should be excised as soon as is possible for fear of sympathetic ophthalmitis occurring at a later date in the other. This may arise at any time between four weeks and possibly even a number of years after the original injury, there are cases recorded which have occurred 10, 15, and 20 years after the original injury. If, therefore, a wounded blind eye is allowed to remain, it is *often* a source of great discomfort to the patient, and *always* a source of danger to the uninjured one. How the infection travels from the exciting to the sympathising organ is not yet settled, but various theories have been suggested:—(1) The infection may spread back along the sheath of the optic nerve of the exciting eye, around the optic commissure, and forward along the sheath of the opposite optic nerve to the other eye. (2) The infection may get into the blood stream, and be conveyed to the sympathising eye through the blood vessels. (3) The condition in the sympathising eye may be brought about by reflex irritation through the ciliary, or optic nerves. It is extremely important to recognise the very earliest symptoms of *sympathetic irritation* in what was previously looked upon as a sound eye. The important symptoms are mainly photophobia with some slight lacrymation, accompanied by sharp neuralgic pains in the eye, and often referred to the forehead, and even just below the orbit. The patient's vision becomes dimmer, and he may have subjective colour sensations. The pupil soon becomes contracted, and dilates somewhat sluggishly when covered, and there may be seen a red zone of slight but deep injection around the margin of the cornea. At this early stage the exciting cause should be *at once removed*, otherwise true sympathetic ophthalmitis will follow, and the patient will then run a great risk of becoming totally blind in the sympathising eye as well as in the exciting eye. Sometimes an eye, affected with sympathetic ophthalmitis, becomes quite blind and later painful. It should then of course be removed.

Preparations, Inventions, etc.

MIOL.

(Miol Manufacturing Co., Ltd., Southwark Bridge Road, S.E.)

"Miol" is an iodo-phosphated oleo maltose diastase, and is composed of the finest Provence olive oil, the active principle from seaweed, in a free and genus form, phosphorus (free), maltose diastase, and the essential properties of Jordan almonds. It is not only a good substitute for cod-liver oil, but it supersedes it by possessing several distinct advantages over it. It causes no gastric irritation, does not give rise to nausea, is palatable and pleasant to the taste, and is economical. Its value as a flesh-forming food, and as a therapeutic remedy, has now been firmly established, and it is specially indicated in cases of wasting, early phthisis, anæmia, malnutrition, and also in cases calling for treatment by iodides, whooping cough, bronchitis, etc. Its especial distinction is that it makes flesh, bone, and muscular but not adipose tissue.

(Chas. H. Phillips Chemical Co., 14, Henrietta Street, W.C.)

Milk of Magnesia.—This is a hydrated oxide of magnesia ($Mg H_2 O_2$), and is a very safe form for the administration of magnesia. Each fluid ounce represents 24 grains of hydrate of magnesia. It is specially useful as an antacid and corrective for excessive acid conditions of the stomach and intestines, as it is free from the many objections pertaining to other forms of magnesia. Its fluidity, perfect hydration, and strong affinity for acids render it completely soluble in the gastric juice, thus precluding formation of intestinal concretions. It combines advantageously with colchicum, iodides, or salicylates in the treatment of gouty, rheumatic, or lithemic conditions. It is also specially useful for the correction of oral acidity, from its remarkable capacity to neutralise an acid saliva. As a mouth wash, it is valuable from its ability to maintain its alkalinity for a long period, and from its destructiveness to micro-organisms.

Wheat Phosphates (Acid).—This is a new preparation of the soluble phosphates which are contained in wheat, and is a nutrient tonic without alcoholic stimulant, capable of relieving mental and physical prostration. Each fluid drachm contains

2 grains of phosphoric acid, 1·75 gr. potassium phosphate, 1 gr. magnesium phosphate, ·75 gr. calcium phosphate, ·2 gr. ferric phosphate, and traces of sodium phosphate and sulphuric acid. It can be highly recommended in cases of impaired mental activity, anæmia, temporary embarrassment of nutrition, nervous exhaustion, etc. It may also be given in cases of incipient phthisis, and is useful as a nutrient in convalescence from typhoid and other fevers. As it contains no sugar, it may safely be given in conditions in which saccharine matter is contra-indicated.

Phospho-Muriate of Quinine (*Compound*).—This compound contains, in each fluid drachm, $\frac{1}{4}$ gr. muriate of quinine and $\frac{1}{120}$ gr. strychnia, in addition to the components of wheat phosphates as shown above, and simple syrup, and is a reconstructive tonic, pleasant to take. It is indicated in the subacute and chronic conditions of disturbed nutrition as expressed by anæmia, tissue waste with neurotic symptoms, catarrh of the respiratory tract, atonic states of the gastro-intestinal canal, etc., and is particularly useful during convalescence. These preparations can all be obtained in quantities suitable for dispensing.

TABLOIDS AND SOLOIDS.

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"*Soloid*" *Products for Ophthalmic Use*.—These products are both valuable and economical in the preparation of solutions for ophthalmic purposes, as will at once be seen from the following details:—

"Soloid" Atropine Sulphate, grains ·545.
"Soloid" Atropine Sulphate, grains ·272, and Cocaine Hydrochlor., grains 1·09.
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These are supplied in tubes containing 6 each.
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"Tabloid" Ophthalmic Physostig. Salicyl., grain $\frac{1}{2000}$.
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These new strengths of these products are now issued. Placed on the conjunctiva they readily dissolve, and quickly produce their effect.

THE PRACTITIONER.

JUNE, 1907.

VALVULAR DISEASE OF THE HEART.

VI.—TRICUSPID REGURGITATION.

By RAYMOND CRAWFURD, M.A. Oxon., M.D., F.R.C.P.,
Physician to King's College and the Royal Free Hospital.

TRICUSPID regurgitation exists whenever, during systole of the right ventricle, some of the blood, that should pass forward into the pulmonary artery, passes back into the right auricle. It is by far the most common of all valvular defects, though less often identified in clinical records than mitral regurgitation. In fact, few cases of mitral regurgitation run their full course without the accession, sooner or later, of tricuspid regurgitation. There is, however, this essential difference between regurgitation at the two auriculo-ventricular orifices, that whereas mitral regurgitation is always a pathological phenomenon, tricuspid regurgitation is not infrequently a physiological safeguard. As with the mitral, so with the tricuspid orifice, two agencies co-operate in closure, contraction of the muscular sphincter and adaptation of the valve curtains: consequently regurgitation may be of two types, either *organic* and due to structural disease of the valves, or *functional* and due to muscular incompetence. At the mitral orifice, structural disease is common and functional incompetence rare, while at the tricuspid orifice, structural disease is relatively rare and functional incompetence very common. It has long been recognised that the lesser circulation has the power of accommodating itself automatically to transitory variations of pulmonary blood-pressure, which inevitably expose it to sudden and undue strain. Were it not for this automatic mechanism, there would be constant risk of the force spending itself backward and causing paralytic over-distension of the right ventricle, or forward with rupture of the pulmonary capillaries. In violent exertion, for instance, the right ventricle does, to a certain point, accommodate itself by dilatation to the increased call, but, beyond this point, relief is sought from the strain by the safety-valve action of the tricuspid sphincter, permitting a leak. If, with healthy valve apparatus and a normal ventricle, incompetence is an established entity, it suggests that the muscular sphincter must be a very important agency in the closure of the orifice. Such transitory functional regurgitation disappears with the need that called it into existence, and is an expression of healthy rather than of morbid action.

In the causation of tricuspid regurgitation of *structural* origin, we have to do with the self-same causes as are operative at the mitral orifice, but whereas inflammatory lesions are far more common on the left side of the heart, degenerative changes are commoner on the right. Acute endocarditis is indeed a relatively infrequent cause, though, as we have shown, in discussing tricuspid stenosis, it is by no means so rare as is commonly supposed, resolution occurring much more readily under the low pressure of the pulmonary circulation. To the same circumstance may be ascribed the infrequency of its occurrence, the location of left-sided lesions affording abundant evidence of the rôle of traumatic influences in exciting acute endocarditis. Structural disease of the tricuspid orifice of inflammatory origin, if it persists, is presumably evidence of an intense and widespread endopericarditis in a large majority of cases, for with it is almost invariably found disease of the aortic and mitral valves, and very frequently of the pericardium as well. It is in this group of cases, with the likelihood of adhesion of the free borders of the valves, that we are most likely to find regurgitation associated with stenosis. Rheumatic fever is, as elsewhere, the usual exciting cause.

It is frequently asserted that the tricuspid valve is the most common site of a malignant infective endocarditis. As a fact, it is far less often so than the mitral valve, and one would expect this, for such a process seldom arises, except in a previously diseased valve of low resisting power.

Rupture of a tricuspid valve is of the very rarest occurrence. It has not the unyielding character, as we have seen, of the mitral or aortic valves, and the readiness, with which it allows reflux under stress of undue strain, is a safeguard against aceration, or rupture of a chorda tendinea.

Degenerative lesions of the tricuspid are relatively common. They frequently follow in the train of disease of the mitral valve, as a consequence of persistent high pressure. Mitral stenosis, as one would expect, is a more frequent antecedent than mitral regurgitation, because the former, as a rule, throws more strain on the right heart than the latter. Mitral regurgitation is more apt to be associated with tricuspid incompetence due to dilatation of the orifice. We are also apt to find hardening of the tricuspid valve associated with chronic diseases of the lung, and with arterio-sclerosis, or granular kidney, and these latter, even in the absence of mitral stenosis, or of breakdown of the left ventricle. Far more commonly, however, these conditions lead to functional incompetence without structural change in the curtains of the tricuspid valve. Among chronic diseases of the lungs, those that bear most hardly on the right ventricle are emphysema, asthma, and conditions of fibroid induration. The full burden of these diseases falls on the pulmonary circulation, obstructing it in the carriage of blood in the pulmonary arterioles, and in the return of blood by the pulmonary veins to the left auricle.

Tuberculosis of the lungs, unless of fibroid type, does not commonly produce this effect. Diseases of the bronchi, primarily at any rate, exert their chief influence on the bronchial circulation, which has its origin directly and indirectly in the thoracic aorta, but the connections between the greater and lesser circulation in the lungs are intimate, and, sooner or later, even in the absence of emphysema, the effects are felt in the right ventricle and on the tricuspid valve.

One might expect that pulmonary stenosis of high degree should likewise lead either to structural, or functional tricuspid incompetence ; but this, I fancy, is very seldom the case. In the first place pulmonary stenosis is commonly of congenital origin, and finds relief in patency of the septum on one or other side of the tricuspid valve, and further subjects of advanced pulmonary stenosis usually die early in life from intercurrent disease of the lung.

It is noteworthy that conditions of the left heart and lungs, which so commonly induce tricuspid incompetence, only, in rarest instances, produce incompetence of the pulmonary valves. This is chiefly due to the firm ring of insertion of the pulmonary valves. There is, however, a degree of high pulmonary pressure, as we have noted in some cases of mitral stenosis, to which even this yields. Apart from incompetence, we do not even find hardening of the pulmonary valves, no doubt due to the ready yielding of the tricuspid; we are too apt to regard the valves as the doors of a series of water-tight compartments, whereas, in reality, they are no more than partial stops periodically interposed in a circuit that is continuous and indivisible.

We have so far considered those conditions which exert their effect variously, in producing either structural changes in the tricuspid valve, or incompetence of the valves without structural change; over and above these are morbid states that produce only this latter form of incompetence. Prominent among these are degenerative affections of the myocardium, transient as in the febrile state, or abiding as in fatty or fibroid degeneration, with or without adherent pericardium. The effects of adherent pericardium are so overwhelmingly more felt on the right side of the heart that tricuspid regurgitation, without obvious cause, should always suggest the possibility of its presence. Profound states of anæmia quite often exhibit, clinically, unmistakable evidence of tricuspid regurgitation with typical backward pulsation in the jugular veins. Here, no doubt, we have to do with muscular enfeeblement of the right heart from malnutrition. Several eminent clinical observers maintain that gastro-intestinal and hepatic diseases are apt to be associated with tricuspid regurgitation. It is alleged that this is brought about through the medium of the great sympathetic, causing spasm of the pulmonary arterioles, and that this is more apt to be associated with acute dyspeptic disturbances than with chronic disorders. I cannot claim to have any experience of such a condition which I have sought and failed to find. Nor do I think that chronic disorders of digestion, involving serious and persistent malnutrition, are by any other means often directly productive of tricuspid incompetence. My mental inclination is to look for evidence at the pulmonary orifice, during systole, rather than at the tricuspid.

Functional incompetence of the tricuspid orifice is not simply a matter of stretching of the tricuspid ring, and enfeeblement of its muscular sphincter: one must also take into account the virtual shortening of the musculi papillares and chordæ tendineæ, resulting from the actual dilatation of the right ventricle. Presuming even that the musculi papillares can, and do, undergo some elongation, pari passu, with the stretching of the ventricle, we can hardly expect an equivalent change in the less yielding chordæ tendineæ.

The morbid changes at the tricuspid orifice in structural disease run so closely parallel to those of the mitral valve in mitral regurgitation, that they need only be considered in outline. In recent acute endocarditis, one may find a fringe of small soft vegetations close to the free border of the valve on its auricular aspect. This condition of the tricuspid valve is not infrequently found side by side with long standing disease of the mitral. In cases of malignant type, these are apt to form large crumbling excrescences, not confined to the valve, but involving the mural endocardium. Such a process is nearly always engrafted on some pre-existing chronic disease of the valve, of which traces may be seen, unless deleted by destructive ulceration. The chronic changes, that follow at times in the wake of acute endocarditis, consist of thickening and induration of the valve, and shortening of the chordæ tendineæ, perhaps with fibroid induration of the musculi papillares. In a tricuspid valve, the changes are apt to be less advanced and less uniform than in mitral : quite often the duration is patchy and not uniform, so that the curtain retains considerable pliability. In more advanced cases, the thickening is uniform, and the valve may be deformed by contraction and even calcified. Rigidity and shrinking of the tricuspid valve are much less apt to produce obstruction along with incompetence than is the case with the mitral : the combination of obstruction of high degree with incompetence at the tricuspid orifice is probably always due to cohesion of the cusps. Even with considerable hardening of the valves, the orifice will often admit, with ease, more than the tips of four fingers. I have recently had under my care a case of tricuspid regurgitation in which the orifice admitted a small gloved hand right up to the heads of the metacarpal bones.

Rupture of the tricuspid valve has already been mentioned as a condition of extreme rarity. Of its morbid anatomy, one need only add that, from the few recorded cases, it would seem that the laceration of the curtain itself is more common than rupture of a chorda tendinea.

In functional regurgitation, there may be very great incompetence of the tricuspid orifice without any affection of the valves: the stretching of the orifice is at times so great that no conceivable reserve of expansibility could compensate for the defect. Such incompetence is part and parcel of the dilatation of the right ventricle with which it is always associated; but, when the dilatation is not primarily due to intrinsic weakness of the heart muscle itself, the right ventricle may be hypertrophied as well.

Grey induration of the mural endocardium of the right auricle is of the slightest degree, and is in no way comparable with that seen in the left auricle in mitral incompetence. Dilatation of the right auricle is invariable, but, in cases, in which the incompetence has arisen slowly in the train of chronic disease of the left heart, and less often of the lungs, there may be some hypertrophy as well. Here, again, the changes have not the same uniformity as is the case in the left auricle. In one part, the auricular wall may be thinned out to absolute transparency, and, side by side with this, may be thick strands of muscle fibres; the thickening and thinning are disposed more or less in ridge and furrow.

It is the exception to find the coronary sinus materially enlarged, so that it would seem as though the coronary valve were capable, to a considerable extent, of warding off the strain of back pressure. In spite of this the heart may be distinctly congested, deep purple in colour, and unduly firm, from interference with its many-mouthed venous circulation. The main venous trunks entering the right auricle are stretched and flaccid. This obvious dilatation may extend back along the innominate veins, and even to the root of the jugular and subclavian veins, rendering them bulbous, and ultimately inducing incompetence of their valves. The Eustachian valve apparently affords no protection to the inferior vena cava, which is dilated throughout, except where it passes through the diaphragm.

Tricuspid incompetence is so frequently the result of, or associated with, disease, or weakness of the left side of the heart that the lungs are commonly in a state of passive congestion. The lesion itself must produce hyperaemia of the bronchial veins, and therewith bronchial catarrh. Hydrothorax, too, is a common consequence.

The enlarged liver shows in its size and colour evidence of stasis of bile and blood. The essential histological features are fatty degeneration and atrophy of liver cells. Recent observations have induced me to examine a number of specimens with regard to the disposition of the fat, and I am confirmed in my adherence to the traditional description of its accumulation in the peripheral zone of the liver lobule. Cirrhosis is a superadded feature in cases of long standing, but is seldom prominent except in alcoholic subjects.

The same evidences of back pressure are apt to be found in the other abdominal organs.

Transitory incompetence of the tricuspid valve may, and often does, exist without any symptoms at all; but true morbid incompetence hardly can. The last help to the filling of the arteries, the last hindrance to the overfilling of the veins is gone. Hence the face is habitually pale, and, at times, there is lividity of the cheeks, lips, nose, and ears. Sometimes a faint yellow discoloration gives the skin of the face and body a dirty hue, which the staining of the sclerotics shows to be due to carriage of bile into the blood through the medium of the lymphatic circulation. Œdema of the face is seldom seen, even when the patient is confined to bed. This is, in part, a mere question of gravity, but also the tributaries of the superior vena cava are far better guarded by valves than is the case with the inferior vena cava. Indeed, œdema of the ankles is usually one of the first signs to appear, and, though at first fugitive, ultimately becomes persistent, and mounts upwards till it may involve the legs, abdomen, and back. For all this we must be prepared to find a small proportion of cases in which anasarca is entirely absent. I have observed this more than once in advanced cases of tricuspid regurgitation with large pulsating livers, as though, in different cases, back pressure in the inferior vena cava followed alternative routes. Though anasarca is much more common than ascites, the two quite often occur in one and the same patient, but anasarca is almost invariably the first to appear. Ascites is certainly not most apt to be found with the very large pulsating livers. These seem to me to be most common when the onset of incompetence has been abrupt, and no attempt has been made to stem it; perhaps, in such cases, the yielding of the liver is greater, and affords a sufficient reservoir for effusion. On the other hand, ascites is more common as a late consequence of a succession of breakdowns of the right ventricle. It is conceivable that diastole of a large pulsating liver may exercise an aspiratory influence on the blood in the portal circulation, if we are to regard this as the main channel of effusion in ascites. The large liver may give a sense of weight and fulness, and even of pain, in the right hypochondrium and between the shoulders. It is usually tender to pressure. In order to detect hepatic venous pulsation, the large liver should be held between the two hands, when it may be felt to undergo a general and gradual expansile pulsation ; when this is once felt, it cannot be confused with the sudden shock communicated to the liver by the contiguous pulsation of either the right ventricle or the abdominal aorta. The pulse is systolic in rhythm, and due to reflux of blood into the inferior vena cava, just as jugular pulsation is due to reflex into the superior vena cava. In the case of the liver, there are no valves standing as a bulwark between it and the heart. This being so, it is surprising that hepatic venous pulsation in tricuspid insufficiency is not more common than it is. If all those instances in which the pulsation is communicated to the left lobe of the liver from the right side of the heart were excluded, true hepatic pulsation would be a comparative rarity. A priori, one would expect it to precede the appearance of jugular pulsation, but, from my own experience, I cannot determine this. I can, however, confidently say that I have never seen an instance of true hepatic pulsation in which jugular pulsation was not present as well. It is stated that the pulse is more marked in the left lobe of the liver than in the right, because the left lobe is more in the direct line of reflux. I am by no means sure that the observation is correct, and in the anatomical disposition of the hepatic veins, as they join the inferior vena cava, there is certainly nothing to support it. Pulsation, even in an enlarged liver, will be more readily felt in the left lobe on account of its situation ; so, too, will the false transmitted pulse be most apparent here. On the same grounds, one would expect to find relatively greater enlargement of the left lobe a constant feature in every case of passive congestion of the liver, which certainly is not the case.

Dyspnœa of varying degree is inevitable, increased by exertion, and at rest there may be orthopnœa. Headache, giddiness, and insomnia result from disturbed cerebral circulation; hallucination and delusions are not infrequent as the end draws near.

Congestion of the abdominal viscera provokes a train of functional visceral disorders; atonic flatulent dyspepsia, with associated constipation, is the rule. Hæmatemesis is very rare, as the liver stands as a bulwark between the right ventricle and the stomach. Vomiting sometimes proves most intractable in the later stages, and may induce fatal exhaustion. The explanation of these symptoms is only too obvious in the post-mortem appearance of the stomach. The mucous membrane is thickly coated with tenacious mucus, the veins are deeply engorged, and, in extreme instances, blood may be exuded into the stomach. Hæmatemesis is most likely to occur when congestion of the liver is combined with cirrhosis, and the same is true of melæna, diarrhœa, and hæmorrhoids. Commonly there is no palpable enlargement of the spleen resulting from passive hyperæmia, unless hepatic cirrhosis is superadded, in which case toxic influences also seem to play a part. Large spleen in cardiac disease is usually the result of embolism, or a toxic consequence of malignant infective endocarditis. The urine is habitually scanty and high coloured, of high specific gravity, and albuminous, the amount fluctuating with the strength of the heart, and it often shows hyaline, and granular casts, occasionally bile. The percentage of urea is apt to be high, though its total excretion is diminished.

Præcordial prominence is only seen when tricuspid regurgitation is a sequel of left-sided disease arising in early life. Pulsation may be visible over the whole præcordium, and in the epigastrium. When there is much hypertrophy, the movement of the right ventricle may be communicated to the whole lower thorax; localised pulsation is, at times, seen over the conus in the second and third left intercostal spaces. The area of cardiac dulness is increased laterally both to right and left, and the apex is displaced outwards. It is remarkable that, as a rule, there is so little raising of the limit of cardiac dulness in chronic cases of tricuspid regurgitation. In acute dilatation the most conspicuous feature is upward enlargement of the right ventricle, so that the dulness approaches the clavicle; this is very frequently met both in pneumonia and pericarditis. It would seem as though, in acute dilatation, the predominant enlargement were in the infundibular portion of the right ventricle, for the upward enlargement is, at first, out of all proportion to the lateral extension to the right. In chronic cases, much of the lateral dulness is due to subsequent dilatation of the right auricle, but it is not the presence of wide dulness to the right so much as its absence in an upward direction in chronic cases that requires explanation.

In about a quarter of all cases of persistent tricuspid incompetence, a systolic murmur may be heard over the lower part of the sternum at the level of the fourth chondro-sternal articulation, or thereabouts. Functional incompetence, as the result of exertion, is not generally accompanied by a murmur. If one may speak of murmurs in dimensions of space, I should describe it as short and broad, relatively to other murmurs. It accompanies the first sound, never completely effacing it, and is soft and blowing in character. I can only recall one tricuspid systolic murmur that could be called harsh, and, curiously, that was where one would least expect it in the case, to which I have already alluded, and in which the tricuspid orifice admitted almost the whole hand. The murmur is more apt to be present when the valve is diseased than when the incompetence is purely functional, but I am not sure that it is harsher. It is peculiarly localised, being seldom traceable for more than an inch or so in any direction from its point of production; the loudest and fullest murmurs are not propagated as far as the apex of the heart. There is no constant modification with posture, but my impression is that, as a rule, the murmur is more intense in the recumbent posture. The murmur often disappears in functional cases, as the heart regains power and the incompetence becomes less, under the influence of rest and digitalis. As soon as tricuspid regurgitation is free, pre-existing accentuation of the pulmonary second sound will be more or less obliterated. If incompetence is slight, or the right ventricle strong, it may still be louder and clearer than normal.

Cases are recorded in which a systolic thrill has been felt in the tricuspid area, but such are outside my own experience.

I have already alluded to hepatic venous pulse; it remains to consider the systolic venous pulsation in the jugular veins, which is more frequent, more obvious, and more readily studied. It is sometimes forgotten that there may be visible pulsation in the jugulars without tricuspid regurgitation. The respiratory movements, for example, cause a slight swelling up during expiration, and a subsidence during inspiration. Normally, these movements are not appreciable, but, in forced expiration, such as is seen in asthma or whooping-cough, these effects are at once thrown into prominence. Normally the jugular veins are inconspicuous, but, in heart disease, as soon as their emptying is hindered by stasis in the right auricle, they at once swell up, and may stand out as prominent bluish cords beneath the skin. This condition is often seen without any pulsation, exclusive of the normal respiratory movements, for the venous valves may still be competent. Moreover, even when tricuspid regurgitation is undoubtedly present, there is quite often no true systolic venous pulse: for its production the valves that guard the cardiac ends of the jugular veins should have yielded. Also a certain strength of ventricular systole is necessary to show it up, and it is the absence of this, in many cases of tricuspid regurgitation, that makes the phenomenon seem relatively infrequent. With a vigorous ventricle, the pulsation may be so full and abrupt as to be mistaken for pulsation of the underlying carotid artery. In an early stage of tricuspid regurgitation, it is at times quite easy to see, as one watches the veins, a low wave due to systole of the auricle, followed by a fall due to auricular diastole, and a higher wave due to ventricular systole, followed by a second fall due to diastole of the ventricle. When, however, regurgitation is free, and the strength of the right auricle completely spent, the pulsation loses its double movement, and reflects only ventricular systole. It may be impossible, by the naked eye, to distinguish this from the single auricular pulse that is seen in cases of tricuspid stenosis, and also in cases of mitral back-pressure, in which the right auricle is hypertrophied, and the tricuspid valve still competent.

At times, one may see an intermediate condition, in which the pulsation is limited to the so-called jugular bulbs. Here all the cardiac conditions of fully-developed jugular pulsation are present, but the valves near the lower ends of the jugulars are still competent; the result is, that their lower ends become distended into domed venous sacs, which may be seen rising and falling above the clavicles, especially when the head and shoulders are low.

Jugular pulsation is commonly more marked on the right side of the neck than the left, because the right innominate vein is more in a direct line with the superior vena cava than is the case with the left innominate.

The diagnosis then of tricuspid regurgitation depends, for the most part, on the presence of a systolic murmur in the tricuspid area, together with such venous pulsation as I have described in the liver or jugular veins. The venous pulse is a more valuable criterion than the murmur, for, even when present, it is often very difficult to decide its site of production. Again, when regurgitation occurs simultaneously at each auriculo-ventricular orifice, it may be quite impossible to assert the existence of an independent tricuspid murmur, as the mitral murmur may be propagated throughout the tricuspid area, and will tend to overpower the short and soft tricuspid systolic murmur. The appearance of dropsy in the systemic venous territory does not necessarily indicate tricuspid regurgitation, and does indeed occur in low degree, while the valve is still competent. High degrees of back pressure, in the absence of tricuspid stenosis, almost necessarily do. Similarly one may find considerable enlargement of the liver while the tricuspid valve is competent, but hardly ascites, or other signs of grave portal stasis of cardiac origin, without incompetence, or obstruction of the tricuspid orifice.

The outlook in tricuspid regurgitation is of very varying moment according to the nature of its cause. If of accidental, of febrile, or of gastro-hepatic origin, it is essentially temporary, and will disappear with rest and attention to the digestive functions, and, at the same time, the signs and symptoms of loss of circulatory equilibrium will be of the slightest. If due to disease or essential weakness of the heart muscle, to adherent pericardium, or to chronic lung disease, the incompetence may disappear for the time, with rest and appropriate medicinal treatment, but will inevitably recur, while the balance is never so completely restored as to abolish all sense of cardiac impairment. At each recurrence, relief is less complete and less lasting, so that the progress is constantly downhill. In such a case, it matters but little whether the incompetence is purely functional, or whether the valve is the seat of organic change as well. Death, in the absence of intercurrent trouble, comes by gradual cardiac asthenia, often with widespread dropsy.

In the direct treatment of tricuspid regurgitation, we have, as in mitral regurgitation, two main lines of action, viz., to diminish the resistance and to increase the power. Rest is the first and most essential agent, and, alone, will often suffice to restore equilibrium ; rest should at first be complete and in bed, and should be relaxed only as improvement becomes marked. The most valuable auxiliary of rest is digitalis, which, beyond all question, acts as powerfully on the right ventricle as on the left. In grave cases, it should be given with the usual precautions, viz., after a free initial purge to unload the veins, and, combined with more rapidly-acting stimulants, such as strychnine, ammonia, or ether, to tide over the delay of its effective doses. Calomel is probably the best purgative to employ, as it seems to be diuretic in conditions of cardiac back pressure. Digitalis should be given at first in small and gradually-increasing doses, so as to give the pulmonary circulation time to readjust itself to the increased driving power of the right ventricle ; not a few instances of hæmoptysis have resulted from disregard of this condition. It is remarkable, as a rule, how, with rest and digitalis, in a few days the circulation is steadied, free diuresis set up, and dropsy begins to disappear. At the same time the murmur and systolic venous pulse may vanish. At times diuresis fails, or is inadequate ; then theobromine or caffeine may be given with salicylate of soda. When the whole body surface, and perhaps the serous sacs as well, is dropsical, these measures are all apt to be quite ineffectual, until such effusion has been relieved or removed ; then paracentesis of the abdomen or thorax, or puncture or incision of the feet and legs may be a necessary prelude to the success of digitalis. Such relief is usually followed by a rapid increase in the output of the urine, and by a return of restful and comfortable sleep. There are yet other cases, and these are commonly those, in which dropsy is not a prominent feature case, in which the strain on the right heart is so great as to render the patient livid, and on the verge of suffocation. Then early venesection, followed by diffusible stimulants, such as ammonia and ether, offers the best chance. Acute distress of this kind in tricuspid regurgitation is nearly always attended by two dangerous auxiliaries, sleeplessness and vomiting, directly due, in each case, to venous stagnation. Sleep must be obtained, and, at times, at any price. I have seen the end, in one or two cases, accelerated by morphia, but far more often by the fear of morphia. I am sure that in conditions of urgent cardiac distress, with restlessness and insomnia, we are far too fearful of morphia, and patients are allowed to die of sheer exhaustion for want of sleep. I fancy it may be given most safely in the form of suppository, but, if given subcutaneously, it may be advantageously combined with strychnine. It is useless to give morphia, if we have not the courage to give an efficient dose. It is idle, in advanced cardiac disease, to put one's trust in hypnotics other than morphia ; the habitual sequel is a night of disturbed delirium, followed by a day of tantalising drowsiness. Trional, sulphonal, and paraldehyde are most commonly employed, because, forsooth ! they do not depress the heart. Paraldehyde has the added horror of a clinging and nauseous after taste, and may excite persistent vomiting.

Vomiting is another grave danger, and is directly due to venous stagnation in the stomach. At times, even the smallest amount of fluid food is at once rejected. In such a state, it is worse than useless to attempt feeding by the stomach. The obvious indication is, without delay, to unload the portal circulation. Any bulky saline draught will be immediately rejected by the stomach, and a full dose of calomel, with an equal quantity of bicarbonate of soda, mixed in a dessert-spoonful of milk, is most likely to be retained ; this will often prepare the stomach to tolerate small amounts of peptonised milk. Rectal feeding is probably useless, as the condition calls for urgent and immediate relief.

It is in this same class of cases, in which general anasarca is not a prominent feature, that the enlarged liver is apt to cause a great deal of discomfort and even pain. Free leeching over the liver, with a brisk purge, may afford some measure of relief.

DUODENAL ULCER.[1]

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DUODENAL ulcer has been considered an extremely rare disease. It was said by Obermayer that its diagnosis is "frequently impossible, or can be made only with more or less probability," and, again, that "the prognosis, provided that the diagnosis can be made at all, is always very serious." These quotations, from the most eminent authority upon the subject, show what the condition of our knowledge of this disease was, at the time, when the surgical investigation of the diseases of the upper part of the abdomen had hardly begun. I hope to show that the duodenal ulcer is common, that its diagnosis in the great majority of cases is quite easy, and that its surgical treatment is the safest, indeed, often the only reasonably safe treatment, that can be adopted.

It is probably no exaggeration to say that, among the advances in our knowledge of the diseases within the abdomen, which have resulted from the more frequent operations in recent years, there is none of greater importance than that which refers to ulcer of the duodenum. In the year 1901, I reported 7 cases of duodenal ulcer upon which I had operated ; a larger number, I believe, than had been reported by any individual surgeon up to that time. In only one of these cases was a positive diagnosis of duodenal ulcer made, for the system-complex was not clearly defined, nor was any help to be derived from the text-books of that day. The descriptions of the clinical manifestations of ulcer of the duodenum were then necessarily imperfect, for the only pathological evidence, making the diagnosis certain, was that afforded by a post-mortem examination. And it was naturally not often that a patient died in hospital, unless one of the acute catastrophes, perforation or hæmorrhage, had necessitated his urgent admission there. When the surgical treatment of the diseases of the gall-bladder and of the stomach became established, it was found that, in some instances where gall-stones had been suspected, the gall-bladder was healthy, but the duodenum diseased, and, in others, that a gastric ulcer was associated with a duodenal ulcer, or that when the former was suspected the latter alone was discovered. And in this way it became recognised, little by little, that ulceration of the duodenum was more frequent than had been supposed, and it was permitted to the surgeon to discover that the clinical history of duodenal ulcer was, in many cases, very easily discriminated from that due to the other diseases I have named ; that the clinical picture, presented by it, was in fact often quite characteristic.

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[1] An address to the Cleveland Branch of the British Medical Association.

Up to the end of 1906, I had operated upon 114 cases of duodenal ulcer ; in 11 of these, the ulcer had perforated ; in 2 cases, the ulcer was associated with a condition of hour-glass stomach. It is upon the records of these cases that the following description of the clinical history of duodenal ulcer is based.

Duodenal ulcer, properly called "peptic" ulcer, may be present in any part of the duodenum, but is most commonly found immediately beyond the pylorus. Collin, in a series of 262 cases, the records of which he had collected, found that, in 242 cases, the ulcer was in the first part ; in 14, in the second part ; in 3, in the third part ; and in 3, in the fourth part. Perry and Shaw, in 149 cases, found the first part affected in 123, the second in 16, the third and fourth in 2 ; in 8 cases the ulcers were scattered. In my own cases, an ulcer has been found in the second part, or at the junction of the first and second parts, seven times, in the remaining 107 cases, in the first part. As a rule, the ulcer is on the anterior surface, and extends up to the pylorus. It may be small, circular, slightly indurated, and cause no stenosis. It may be large, hard, puckered and adherent, and a high degree of narrowing may result from its cicatricial contraction. In a few instances, the ulcer may be so surrounded by an inflammatory deposit that a large, easily palpable tumour results ; in one of my cases, a lump as large as a man's fist was found. In a few instances, multiple ulcers are present ; most commonly an ulcer on the anterior surface is exactly apposed to an ulcer on the posterior wall ; "kissing ulcers" these may aptly be called. More rarely an ulcer in the first part is associated with another, or others, in the third or fourth part.

Duodenal ulcer, as I pointed out some years ago, is not seldom associated with gastric ulcer. In about 40 per cent. of my own cases, this connection is shown. It is not improbable that the gastric ulceration is the primary condition, the duodenal ulcer being secondary, and caused, it may well be, by the digestion of the duodenal mucosa by the hyperacid gastric juice. The intolerance of the mucous membrane of the duodenum to acid contact has been illustrated in the work of Pawlow. It is a very striking fact that the part of the bowel affected by the process of ulceration is that with which the food, as it leaves the stomach, is immediately brought into contact.

Duodenal ulcer is found at all periods of life. T. D. Lister and Spiegelberg have related instances occurring in infants 3 to 4 days old, and "melæna neonatorum," in some cases at least, has been shown to be duodenal ulcer. Henoch, Zerschwitz, and Musser have all recorded instances of ulcer in the new-born. I have seen a case in which an ulcer in a child, 19 months old, almost caused death from hæmorrhage. In this case, the boy, who had previously been subject to occasional attacks of vomiting, when 17 months old, suddenly fainted while taking his food ; he became blanched and cold. A few hours later, a copious blood-stained motion was passed, and melæna subsequently persisted for 4 days. Two months later, a similar attack occurred, but the melæna lasted longer, and the child was desperately ill. The oldest patient in my series of cases was 73 years old ; the average age is a little over 38 years. But in many cases the patients had suffered for several years, so that the age given is merely that at which the symptoms had existed long enough to demand operation. The symptoms may begin at any age ; patients of 60 say that they have suffered for 4 or 6 or 10 years, patients of 50 for 2 years, patients of 30 "all their lives," and so on.

Men are more frequently affected than women. In my own series, there are 76 men and 38 women, exactly 2 to 1. Mayo and Graham give the proportion as 2·5 to 1. The ratio given by other writers varies between 4 and 6 to 1. The reason for the greater frequency in men, recognised by all writers, is difficult to ascertain. At one time, I was disposed to think that duodenal ulcer occurred, with noteworthy frequency, in men who drank beer, especially between meals, a "run" of such cases having occurred in a short period, but the history of subsequent patients has not borne out this supposition. A history of hasty, and perhaps heavy, meals at irregular intervals is not infrequent.

Symptoms.—The symptoms of duodenal ulcer are, in the great majority of cases, sufficient to enable a diagnosis to be made without any physical examination of the patient. The anamnesis is to this effect: After food is taken, the patient is free from pain, the period of an hour or two, which follows a meal, is the best time in the day. At a time, varying from 1½ to 4 hours after the meal, a sense of uneasiness is noticed in the upper part of the abdomen. A burning, gnawing sensation develops, and there is a bitter taste in the mouth, with, it may be, eructations of food or gas, bitter and acid in taste. The pain, which gradually increases, may be relieved, often considerably, by belching, or by pressure. As it increases in severity, it strikes through to the back, to the right of the middle line, and it may radiate round the right side of the chest. As all patients discover for themselves, the taking of food relieves the pain, so that many carry a biscuit in their pockets, or take milk, a dose of an alkaline medicine, or some form of food as soon as the uneasiness develops. It will often be found that a patient names certain hours as those at which the pain is noticed, 11 a.m., 4 p.m., or 2 a.m. These are all a few hours after food. Many patients wake up, after a short sleep, with an intense gnawing pain in the pit of the stomach; and several of these have told me that they keep a few biscuits, or some bread and butter, or a glass of milk at the bedside, and that, on waking, these foods are taken to relieve the pain. In several cases, upon which I have operated, the pain has been more severe than this, has been, in fact, indistinguishable from a mild form of hepatic colic; the patient describes the pain, which comes constantly 2 or 3 hours after food, as a "colic" or a "spasm"; it is not improbable that a spasm of the pylorus is actually present, for such a condition may subsequently be seen during the course of an operation. The pain, it will be noticed, comes on at a time when the patient should be beginning to feel hungry for his next meal; for this reason, the term "hunger-pain," which I suggested in a former paper, seems quite appropriate. The interval of relief, after a meal, varies chiefly according to the character of the food taken. The more substantial the food, the greater the interval of relief. If meat is taken in good quantity, there will be a long period of ease, if only fluid foods are taken, the relief is short, or is not experienced at all. Heavy "indigestible" meals are, therefore, not infrequently taken, in order that the intervals of freedom from pain may be prolonged. Though temporary comfort is given, it cannot be doubted that more serious damage is, in consequence, inflicted upon the ulcer. A medical man, upon whom I operated, found that a heavy meal of beef gave him most relief; when he was "dieted" on fluid foods, he was hardly ever free from pain. In addition to the pain, there is a feeling of great distension, of fulness and tightness, a "blown-out" feeling in the epigastrium, and the patient may develop a habit of eructation in the hope of relieving this most disagreeable sensation. This feeling of flatulent distension in the epigastrium is often very distressing; some patients describe it as the most intolerable of all their symptoms. The appetite is generally good, is, in fact, often better than the normal, if stenosis has not developed. It is not unusual for a patient to say: "I've a good appetite, I can take anything, and I never vomit." If he has given a history of pain, as I have described it, one may be confident that he has duodenal ulcer, without stenosis. Investigation by test meals will show no stasis and some hyperacidity.

After a time, a few weeks, a month or two, the symptoms may gradually improve, and even disappear, to reassert themselves after a longer or shorter interval. The patient will then speak of having "attacks" of a certain duration, coming capriciously, leaving spontaneously. In the intervals of these attacks, he may be perfectly well, suffer absolutely no discomforts, enjoy food, and gain weight. What the periods of latency mean, I do not know, for if a patient, who has suffered long, is operated upon in an interval, the ulcer is as plainly to be seen as ever. It may be that, though the scar of the ulcer is there, the mucous surface has healed over temporarily, and that a recurrence of the symptoms is due to a fresh breaking-down of the ulcer, or possibly to the development of another ulcer.

In the "attack" the patient generally, but not always, loses weight. About one-fourth of the patients are sleek and well-conditioned, and this is due most probably to the excessive feeding in which they indulge, in order to obtain temporary relief from pain. A few of the patients, upon whom I have operated, have been short, very stout men, whom, before my experience of the last three or four years, I should never have supposed to be the victims of duodenal ulcer. Constipation is a frequent symptom in duodenal as in gastric ulcer.

The description of the pain, which I have given, will remind you at once of that which is described as being due to "hyperchlorhydria." I have read, in various papers, of "hyperchlorhydria" or "hyperchlorhydric dyspepsia," as if these were diseases of a special type. I have no hesitation in saying that, in almost all such cases, the patients are suffering from duodenal ulcer. If an anamnesis, such as I have recounted, is given, duodenal ulcer may be diagnosed without hesitation, and an operation be undertaken for its relief. The term "hyperchlorhydric dyspepsia" is one of the medical names for the surgical condition duodenal ulcer. If there are exceptions to this statement, I have not met with them. The "acid dyspepsias" which are supposed to be "functional" are in truth, I submit, generally cases of ulcer in the duodenum.

At a time when our knowledge of duodenal ulcer was based upon the only evidence available, that of the post-mortem room, a statement of this kind would have been received with incredulity, but the experience of the operation theatre is so much greater in quantity, its facts can be so closely scrutinised in connection with the details of the anamnesis, that our knowledge is now more precise than it was ever before. The pathology of the living, which the surgeon has the opportunity of studying, enables us to recognise such conditions as duodenal ulcer in an early stage, at a time when simple and comparatively safe methods can be adopted for their cure. It is this which has brought the accurate diagnosis of abdominal diseases more and more within the province of the surgeon. It is he, who is equipped by his daily work with the knowledge, which is born of a close study of the symptoms, and of the exact pathological conditions, which at that moment are responsible for them. He has not to wait, until the final ruin of the patient is accomplished, before being in a position to study the organic changes which are taking place.

Vomiting is an infrequent symptom of duodenal ulceration, it comes as a constant symptom only when stenosis is present, and when the patient has not yet "taken the measure" of his stomach's capacity. If the ulcer is active, and has not caused any narrowing, vomiting is a rare symptom ; many patients say explicitly that they have "never vomited." When the ulcer is cicatrising, and a high degree of stenosis has resulted. there may still be no vomiting, for the patient has learnt how much his stomach can deal with, and he does not exceed that amount. A patient will not seldom say : "I don't vomit now, but I should do so if I were to take a good meal." Even when a large contracting stomach is seen, where the stenosis is considerable, and of old standing, there may be no vomiting.

An examination of the surface of the abdomen will generally reveal a tender area a little above and slightly to the right of the umbilicus. Dr. James Mackenzie, of Burnley, has shown that the area of tenderness, marked out by pinching the skin between the thumb and finger, lifting the skin away from the muscles, so as to avoid deep pressure, will indicate, as a rule, the position occupied by the ulcer of the stomach, or of the duodenum, which is the active cause of the tenderness. If there is an ulcer of the lesser curvature of the stomach close to the cardia, the skin, in the middle line, or slightly to the left near the ensiform cartilage, will be tender. If there is a pyloric ulcer, the tender point will be lower down near the umbilicus. If there is a duodenal ulcer, the tender point is in the position I have named.

The most serious of the symptoms, which result from duodenal ulceration, is hæmorrhage, which may be manifest as hæmatemesis or melæna. In a few cases, it is the only, or at least the chief, symptom. In four of my cases, the history given was that, at intervals, attacks of "faintness" occurred. followed by anæmia : an examination of the stools showed that melæna was present ; then inquiry was made, as to epigastric pain and distension ; and the usual history was elicited. In one case, the "dyspeptic symptoms" were quite insignificant, and the patient could hardly be convinced of the connection between them and the faintness and anæmia. An operation disclosed a typical duodenal ulcer. In 15 cases in my own series, repeated and increasing hæmorrhage has been the urgent cause of operation. In 10 of these cases, both hæmatemesis and melæna were present, but in all save one the melæna was far more profuse than the hæmatemesis. In four, melæna alone was present, in one hæmatemesis alone ; in this case, blood was present in large quantity in the intestine at the time of operation. In addition to these, bleeding occurred in 27 cases.

As hæmatemesis alone in - - - 8 cases.
As melæna alone in - - - - - 7 ,,
As hæmatemesis with melæna in - - 11 ,,

In the total of 114 cases, hæmorrhage, to an extent easily recognisable by the naked eye, occurred, therefore, in 41 cases ; there were not improbably several others, in which, had a careful search been made, the evidence of bleeding would have been obtained.

Hæmorrhage from a duodenal ulcer, whether manifest as hæmatemesis or as melæna, is a far more serious matter than is the bleeding from a gastric ulcer. It is not necessarily more profuse, but is certainly more apt to continue and to recur, and it is even less under control, perhaps because the ulcer is less accessible, perhaps because the vessels opened are larger, than is the case in ulceration of the stomach. Moreover, when an ulcer in the duodenum is bleeding, the amount of blood, which escapes into the stomach to be discharged in the act of vomiting, is no measure of that which is passing onwards in the intestine to be discharged many hours or days later. The visible evidences of hæmorrhage are, therefore, no criterion of the conditions which are present in the ulcer. The patient may indeed die of hæmorrhage when the amount of blood discharged from the body is of no great quantity. In such a case, and in all cases in which operation is performed for hæmorrhage, the intestine will be found well filled with blood. I have often seen the colon distended and dark blue in colour, by reason of the tarry blood within it. If a gastric ulcer is bleeding, the amount of the blood vomited affords a fairly accurate estimate of the actual loss of blood which is occurring ; in duodenal hæmorrhage such an estimate is quite impossible. During last year I saw two cases of duodenal ulcer, and advised operation. When the patients were sent for, to be admitted to the infirmary, the reply came that they had both died of hæmorrhage. I have, therefore, come by degrees to consider that hæmorrhage from a duodenal ulcer is a symptom of extreme urgency and seriousness ; and that, if it does not soon appear to be ceasing, an operation should be performed without delay. The risks of operation are then probably less than the risks of delay. I find that I have operated upon 15 cases because of continuing hæmorrhage. All the patients have recovered. In all posterior gastro-enterostomy has been performed, and, at the same time, the ulcer has been infolded. By so doing, the vessels supplying the ulcer, from one of which the bleeding is occurring, are secured by the suture, the first line of which is drawn very tight. In no case has there been any evidence of further hæmorrhage. This infolding of the ulcer, which probably closes the duodenum altogether, is a most necessary, perhaps the most necessary, part of the operation, and should never be omitted. It is simpler than excision, far quicker, and equally effective.

The chief variations from this history are found in the cases of duodenal ulcer complicated with other diseases. In approximately 40 per cent. of cases of duodenal ulcer, some evidence of gastric ulcer will be discovered. In the majority of such instances the ulcer in the stomach has ceased to be active, being represented only by a scar, or by a firm localised adhesion to the abdominal wall, the liver, or the pancreas. In two cases in my series, there was an hour-glass stomach, due to old ulceration. If the ulcer in the stomach is active, its activity may be expressed in symptoms which are recognisable. Pain will come at an earlier time after food, the surface tenderness is higher in the middle line, or to the left, along the costal arch, the pain may radiate round the left side, or up to the left breast. In some cases, a very positive diagnosis of the co-existence of gastric and duodenal ulcers may be made.

In seven cases, in my series, cholelithiasis was present, and was dealt with by cholecystotomy, or cholecystectomy. I think it is not improbable that, in my early experience, I may have omitted to observe the condition of the gall-bladder in cases in which it was affected, for I had operated upon a small number of patients before I was brought to realise the connection between the two conditions. In one case, I found a fistula between the gall-bladder and the duodenum, secondary, as I thought, to ulcer in the latter. In another, a hard mass in the duodenum proved to be a gall-stone, which had ulcerated through from the gall-bladder. The duodenum and the gall-bladder were fused together into a mass, which felt like a pyloric growth on external examination of the abdomen.

The only serious difficulty, in the differential diagnosis of duodenal ulcer, is encountered in cases of cholelithiasis. There are cases, very few in number, but not to be ignored, in which the most careful investigation will not permit one to be certain as to which of these two conditions is present. In some patients, who suffer from cholecystitis, as Ewald has shown, there may be temporary marked hyperacidity. Such patients suffer from something akin to the " hunger-pain " just mentioned. Pain comes two to three hours after food, at a time when the inflamed gall-bladder is subject to disturbance by the food passing out of the stomach. The pains are, in character, similar to those felt in duodenal ulcer, they are cramp-like, felt to the right of the middle line, they radiate round the right side of the chest, and pierce through to the back. The tender area on the abdomen is, however, much more marked, as a rule, in cholecystitis, and the tenderness, elicited by pressure on the lower margin of the liver during deep inspiration, at the moment when the gall-bladder impinges on the fingers (" Naunyn's sign ") is quite different from that quite definitely localised tenderness found in the uncomplicated cases of duodenal ulceration.

If the patient is fortunate enough to escape both hæmorrhage and perforation, the ulcer may heal completely. A fibrous knot, or ring, then develops in the duodenum, the lumen of which is narrowed to a greater or less degree. The stomach then becomes dilated and hypertrophied, sometimes to an amazing degree. I have twice seen the stomach so large that its lower border on inflation disappeared behind the pubes. In cases, in which a fibrous stricture exists, the clinical picture is that of pyloric stenosis. A knowledge of the exact site of the stricture, whether in the duodenum or in the stomach, is at this stage not possible; but a close scrutiny of the earlier history, if this is remembered, will almost always give the clue to a precise diagnosis.

Perforation of a duodenal ulcer may occur at any stage in the course of the disease, early or late; it is quite certain, however, that it never occurs without previous symptoms of ulceration having been present. These symptoms may have been slight, perhaps almost negligible, or even neglected, but a reasonably close inquiry will always elicit them.

Perforation may occur suddenly, or slowly; it may be of the three types which I have fully discussed elsewhere—acute, subacute, or chronic.

Acute perforation occurs suddenly. The patient is seized in a moment with an intense, intolerable pain in the belly. He may say that "something has given way" in his body. The pain is agonising, the expression of the patient has suffering written in every line. The abdomen is held stiff, and retracted, respiration is shallow, thoracic only, and an attempt to speak is cut short by the inability to take a deep breath. But collapse, which I formerly described as an early symptom, is certainly not present. In the cases I have recently seen (two within a couple of hours of the perforation), there has not been any collapse, the pulse has been slow, not more than 80, and of fairly good quality. It is important to recognise that the conditions, upon which we formerly depended to make the diagnosis, should not be allowed to develop, the distended abdomen, the poor, rapid pulse, the shock or collapse are not the early evidences of perforation, they are symptoms not of perforation, but of the consecutive peritoneal implication. The surface of the abdomen is tender, often exquisitely so, and the tenderness is always more pronounced upon the right side.

A positive diagnosis of perforated duodenal ulcer can, I think, be always made in the few hours following the rupture. The history of the pain at some time after food, of pain relieved by food; the history of the sudden onset of agonising pain; the restriction of thoracic movement, and the impossibility of a deep breath being taken; the retraction of the abdomen, and its rigidity; the increased resistance, and the greatly increased tenderness on the right side, will almost preclude the possibility of a mistake. The skin of the abdomen, when pinched up lightly, will reveal its most tender spot on the right of the middle line above the umbilicus. It is important that the differential diagnosis between gastric and duodenal perforations should be achieved, in order to enable the incision to be made in such manner as to afford the easiest access to the seat of trouble. By careful investigation, the position of a rupture in the stomach, or in the duodenum, can be made with reasonable accuracy, and the abdomen opened to left or right of the middle line accordingly. When the perforation has been left unrecognised for some time, peritonitis on the right side of the abdomen may develop to a degree which, as I showed some years ago, had caused a false diagnosis of appendicitis to be made in approximately 40 per cent. of the then recorded cases. The treatment of a perforating ulcer is immediate operation, and closure, with or without excision of the ulcer, of the opening. If the sutures are put in so as to leave a vertical wound, there may be no narrowing of the lumen, but even so, it is probably better in all cases to perform gastro-enterostomy.

I find that I have operated upon 11 cases of duodenal perforation, with two deaths. In four cases, gastro-enterostomy was performed at once, three patients recovered, and one died (this was an operation done seven years ago, and I used a Murphy button). Of the six patients who recovered without gastro-enterostomy, two have since been compelled to have this operation performed on account of duodenal stenosis. All the patients who recovered are now quite well.

Three cases, which I have had under my care, are worthy of special comment. In one case, that of a man of 40, the ulcer, which had caused symptoms for two years, ruptured when a heavy barrel fell on the abdomen. In the case of a woman, aged 40, a gastric ulcer perforated; it was closed by suture, and all went well until the 11th day, when the duodenal ulcer perforated, and caused death. In a third case, a man of 27 had suffered from vomiting for several months.

A duodenal ulcer perforated. At the operation, a small ulcer, in a much distended duodenum, was found, and the opening closed by suture. When posterior gastro-enterostomy was performed, it was discovered that the first loop of the jejunum was engaged and strangulated in the sac of a right duodenal hernia; the small piece of jejunum outside the neck, and the duodenum, were both distended as a result of the intestinal obstruction. The rupture of the ulcer was probably secondary to the strangulation of the right duodenal hernia.

Among the rare complications of duodenal ulcer, carcinoma may be mentioned. I have once operated upon a patient, who had a history of long-standing duodenal ulceration, in whom a carcinomatous tumour of the duodenum was found. The patient, after gastro-enterostomy, lived for 19 months, and died, after having suffered from jaundice and ascites, with secondary growths in the liver.

Treatment.—The first question which requires consideration, assuming the diagnosis of duodenal ulcer to have been confidently made, is concerned with the value of medical treatment. In cases of gastric ulcer, in an early stage, I have no hesitation in saying that medical treatment is the only treatment to be considered; provided that is thoroughly carried out over a sufficiently long period, the probabilities are that the ulcer will heal. In cases of duodenal ulcer, I do not think that the same statement holds good. An ulcer of the stomach, at least in its usual position, is perhaps not brought into the same intimate contact with food as an ulcer in the narrow duodenum. At the moment when the pylorus relaxes, and food is passed through it, an ulcer, if in the common position in the duodenum, must be encountered. Healing of the ulcer is less likely to occur, and if healing should occur, there is surely a greater liability to breaking down of the thin scar tissue, under the constant irritation and friction to which it is exposed. In the cases which I have seen, both hæmorrhage and perforation have occurred more frequently in duodenal ulcer than in gastric ulcer. Medical treatment, however carefully carried out, has proved of less value in duodenal ulcer than in gastric ulcer. My view, therefore, of duodenal ulcer is that it is a far more serious disease than gastric ulcer; that hæmorrhage is more likely to occur; that, when it does occur, it is less amenable to treatment ; that perforation is mo prone to happen, and that a more serious operation (owing to the more frequent need for gastro-enterostomy) is then necessary ; that medical treatment is of far less value ; and that relapses are proportionately more frequent.

For these reasons, I have come to the conclusion that the question of surgical treatment should be entertained at an early period of the disease. If the diagnosis is certain, then I think that surgical treatment is safer, and more certain than medical treatment. If hæmorrhage has once occurred in even moderate quantity, surgical measures should be adopted as soon as possible, for the future of a patient, in such circumstances, is precarious ; at the best he is almost certain to suffer serious discomfort in longer or shorter "attacks," and, at the worst, he may die from perforation or uncontrollable hæmorrhage. I have, for the purposes of this paper, looked through the records of all the cases of duodenal ulcer, upon which I have operated ; excluding the cases of perforation, there are 101 cases of duodenal ulcer, which I have treated by operation. Posterior gastro-enterostomy has been performed in all, by the method which I have described elsewhere ("Abdominal Operations"). In the majority this has been combined with the infolding of the ulcer, by a double layer of sutures. In this series, there have been two deaths. The after-results are extremely good, in only one case has a bad result to be recorded. This patient had to be operated upon a second time by another surgeon, who found evidences which led him to believe that a peptic ulcer had developed in the proximal part of the jejunum.

Five patients cannot be traced, so that details of their recent history are lacking. Three patients are to be classed as "improved" by the operation ; all suffer at times from flatulence, fulness after meals, occasional vomiting, and acid eructations. The remainder are all to be classed as "very good" results, the patients are fully restored to health, and most of them are better than for years before the operations. The results of the operative treatment of duodenal ulcer are, therefore, extremely satisfactory.

It has long been my custom, in all cases of duodenal ulcer, treated by operation, more especially in those in which there has been excessive hyperchlorhydria, to advise that a strict dietary be followed for three months after the patient returns home, and that an alkaline bismuth mixture be taken, if there is any indication of persisting acidity.

The reason for this is as follows:—The new opening between the stomach and the jejunum replaces the pylorus. As food passes through this opening into the jejunum, the mucosa of the bowel is submitted to the same conditions as was the duodenal mucosa before the operation; and, as a result of these, similar changes may occur in it, leading to a peptic ulcer of the jejunum. Such a peptic ulcer has been observed in over 30 recorded cases, and in all has proved to be a serious complication. What the cause of jejunal ulcer is has not been definitely ascertained, but some attention to the careful dietary of the patient is desirable in order to prevent anything of the kind occurring. It will be constantly found that the rapid return to robust health of a patient, upon whom gastro-enterostomy has been performed, is attended by an astonishing increase in his appetite, which becomes almost voracious. The keen zest for food in one, who has long paid a penalty for each meal, leads to over-eating or over-drinking. It is, therefore, most desirable to warn patients that a judicious restraint is necessary for a few months. Almost all patients gain weight rapidly, and, in one remarkable case of mine, the patient's weight was doubled in 12 months (5 st. 2 lbs. to 10 st. 6 lbs.).

Such, then, very briefly put, is my experience of duodenal ulcer. The fact that one surgeon has operated upon 114 cases disposes of the statement that duodenal ulcer is rare; on the contrary, it is frequent. Its supposed rarity was due to an inability on our part to make an accurate diagnosis; the living pathology had not been studied to a degree permitting us to make the diagnosis with such certainty as is now possible. The disease, moreover, has long masqueraded under many other names, "hyperchlorhydria," "acid gastritis," "acid dyspepsia," and so forth. Finally, we are, I venture to think, entitled to say that surgical treatment has proved to be of such value, and of so comparatively slight a risk, that its advocacy, in an early stage of the disease, is in the best interest of the patient.

THE PRETUBERCULOUS STAGE OF PULMONARY TUBERCULOSIS.

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The importance of the early diagnosis of pulmonary tuberculosis need not now be emphasised, as it has come to be recognised that, on the early detection of the disease, depends in a large measure the success of treatment. In the modern conception of pulmonary tuberculosis, the so-called pretuberculous stage is coming every year to occupy a larger share of attention. The pretuberculous stage of the disease may be roughly said to be that early stage, at which the physical signs are indefinite and insufficient of themselves to justify a diagnosis of pulmonary mischief. But these indefinite early signs are associated with a condition of ill-health, which should arouse suspicion as to the probable causal factor. They constitute a "clinical picture" which, like all clinical pictures, can only be recognised by experience. In the present paper, I purpose alluding to some of the ailments which occur during this stage, and to some of the more modern methods, which may be employed to clear up the diagnosis.

Pulmonary physical signs have been the subject of much debate in medical literature. The question as to how much deviation from the normal, or supposed normal, is necessary to clinch a diagnosis of lung mischief, and the relative values of auscultation, percussion, and X-rays, in determining the presence of disease have no doubt their importance. Over-attention to these tends to exclude from consideration certain other factors which are of undoubted use in arriving at a diagnosis in obscure cases.

One of the questions which has not received, in this country at least, the attention it deserves, is that of the time which elapses between the date of infection and the actual onset of what are at present recognised as the early signs of the disease. In many cases, and by many observers the question is ignored; the actual knowledge that tuberculosis is the result of the introduction of the specific organism into the system is not applied to the individual case.

The solution is obscured by many difficulties. First in importance, I may mention the length of time which elapses between infection and the development of symptoms, and the absence of any primary and easily recognisable lesion. The difficulty is further increased by the fact that the pulmonary is probably rarely the primary lesion. The consequence is that writers, who attach importance to infection, are divided into two schools—those who believe that an attack of tuberculosis is referable to a definite infection, and those who hold that the great majority have in their systems latent foci which lie quiescent for an indefinite length of time till lighted up by some intercurrent disease, or other depressing influence lowering the general resistance.

The arguments, adduced in favour of the two apparently opposing conclusions, are well summarised in Dr. Burton Fanning's excellent book on the open-air treatment[1] under the heading of "Ætiology. He quotes the researches of Niven, who, in 1,006 cases, found distinct evidence of infection in 64·8 per cent., and Hilier, who holds that, in the majority of cases carefully recorded, there is found a history of infection. On the other side, he cites the work of Birch-Hirschfeld, who found evidence of the presence of the tubercle bacillus in 40 per cent. of 4,000 necropsies, and of Naegeli, who states that tubercle is discoverable somewhere in the bodies of all persons who have passe the age of 30.

I think that the two explanations of the onset of the disease are reconcilable. It is probably true that the great majority of those, who have passed early life, have latent foci of disease, and that these foci may become active from a depression of the vitality and intercurrent disease. But, on the other hand, the repeated absorption of virulent bacilli into the system is the surest way of depressing the specific resistance to tuberculosis. Then, even supposing that the latent focus may contribute to the establishment of a more general active condition, the repeated and prolonged exposure to infection, which almost invariably occurs in those cases in which a history of infection is demonstrable, is the real cause of the lowering of resistance and the starting-point of active mischief. In the remaining cases, infection may not be actually demonstrable, but none the less it may have occurred, considering the almost omnipresence of the germ under modern civilised conditions, and, by lowering the resistance, it may light up a quiescent focus, or start a fresh area of disease, this being assisted in many cases by intercurrent debilitating conditions. If we follow out the course of infection, we are still further impressed by the difficulty of diagnosing from physical signs. The primary focus may be in the tonsil, the pharyngeal lymphoid tissue, cervical lymphatic glands, or in the bronchial glands. The lesion in the bronchial glands may be secondary to any of the others, or, if primary, may be followed by spread into the lung tissue itself. The spread into the lung tissue takes place along the line of least resistance.

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[1] Burton Fanning: *Open-air Treatment of Pulmonary Tuberculosis*, pp. 13, *et seq.*

Birch-Hirschfeld[1] shows that the initial lesion occurs at the bifurcation of a particular bronchiole in the posterior part of the apical lobe, and not at the extreme apex where the lung is readily accessible to the stethoscope. This occurs at the right apex with about the relative frequency of 3 to 2.

Woods Hutchinson[2] argues that the right apical lobe is most frequently affected because it is undergoing ancestral degeneration, and is therefore more unstable and liable to disease. The argument is ingeniously worked out in his book on comparative pathology, in which he shows the fallacies which underlie some of the more familiar explanations such as deficient expansion. The lesion in the bronchial glands, and, to a less extent, the early pulmonary lesion are out of reach of the stethoscope, and obscured from the X-rays. The condition, when certainly demonstrable by the stethoscope, does not represent the earliest ravages of the disease, and still less corresponds with the lowering of resistance which is the start of the trouble.

Dr. Burton Fanning[3] well emphasises the difficulties underlying the true interpretation of physical signs:—"As regards the extent of the lesion, the evidence of the physical signs is fairly clear and trustworthy, always allowing for the fact that they only bear witness to the more superficial and grosser changes. There will be deposits of tubercle of which no indications are given by auscultation and percussion, and in the more central portion of the lungs even breaking down may occur without the possibility of detection." If the above is true with reference to well-established disease, how easy it must be to fail to observe a deeply-seated early small focus.

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[1] Birch-Hirschfeld: *Deutsche Archiv. f. Klin. Med.*, Vol. 64.

[2] Woods Hutchinson: *Studies in Human and Comparative Pathology*, 1901, p. 80, *et seq.*

[3] Burton Fanning: *loc. cit.*, p. 60.

If the earliest appearance of physical signs at the surface does not indicate the commencement of the initial lesion, the question arises as to how long the process has been in operation before detection becomes possible. On this point we have extremes of opinion, from that of Behring, who believes all tuberculosis to be secondary to infection through the ill-developed intestinal mucosa of the child, to that of the many, who date the commencement of the mischief from practically the commencement of unmistakable disease.

Enquiry into the past history, even of the earliest cases, reveals a period of ill-health stretching back for months or years, during which the causal pathological condition has been progressing, culminating in the development of the condition recognised as early pulmonary tuberculosis. Ruata[1] states that he has been able to obtain a history of definite exposure to infection in as many as 90 per cent. of his cases. He finds that the average period, which elapses between exposure to infection and the onset of definite physical signs, is from 2–4 years. I have been able to get decided evidence of infection in only 31 per cent. of 150 cases. In many of these cases, the infection was conveyed from a relative, or from someone working at the same business. In these cases, I have calculated the date of exposure to infection from the death of the person from whom the infection was conveyed to the date of diagnosis. I found it to average 3 years 2 months. Taking the death of the person as the starting point obviously unduly shortens the period of invasion, and infection, or exposure, was in the great majority of cases prolonged and repeated, and began long before the date taken, but it at least gives us some indication of the length of time. It will thus be seen to be 3 years or more. During this period the disease has spread from the bronchial glands far enough out to be capable of detection. The "latent" disease has gradually taken hold during this "pretuberculous" stage, and there have occurred various signs and symptoms, the direct result of the toxic action of the tubercle bacillus.

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[1] Ruata: *Medical Annual*, 1901, p. 534.

In organismal disease, such as typhoid, in which the initial lesion is deep seated, the diagnosis is arrived at by consideration of the temperature, headache, nose-bleeding, etc., which are the result of the deep-seated process. We come to a diagnosis in such cases by the general grouping of secondary symptoms, and more scientifically by the direct observation of the resisting power of the patient's serum. It is of such a summing up and grouping of symptoms that I would urge the importance in the detection of "pretuberculous" disease.

During this period preceding diagnosis, we find that, although the insidious nature of the disease prevents the patient from realising that he is suffering from a serious illness, and nothing sufficiently alarming occurs to induce him to consult his medical adviser, he has almost invariably been in poor health. He suffers from various functional disorders of his physiological systems. I find, for example, that almost all my cases suffered, previous to diagnosis, from anorexia or want of appetite. The appetite may be fair, but almost never up to the proper standard of good health, and is usually associated with a distinct degree of constipation and more or less marked dyspepsia. The following table shows the percentage incidence of various symptoms prior to diagnosis in 150 cases. Some of them are purely functional disorders, others are more complex, and are classed as separate diseases, usually regarded as having an ætiological relationship to tuberculosis.

Table (150 consecutive cases) *showing the frequency with which patients suffer from various symptoms, etc., during the pretuberculous stage* :—

Anorexia	- in 98 p.c.	
Dyspepsia	- „ 83 „	
Constipation	- „ 60 „	
Hæmoptysis	- „ 26 „	Recurrent in 35 p.c. of these.
Pleurisy -	- „ 46 „	„ „ 3 „ „ „
Liability to colds	„ 38 „	„ „ all.
"Influenza"	- „ 51 „	„ „ 35 p.c. of these.
"Bronchitis"	- „ 10 „	„ „ 71 „ „ „
"Inflammation"	„ 8 „	„ „ 20 „ „ „

In the last three lines of the table the diagnosis is that given at the time of the attack. Similar statistics were worked out by Burton Fanning in relation to previous tuberculous attacks.[1]

While it is certain that conditions, such as influenza, occur during this stage as intercurrent diseases, still, in my own mind, I am equally certain that all conditions grouped as "Influenza" are not really such. True influenza, caused by the bacillus of Pfeiffer, may be one of the causes of lowered vitality which permits of the spread of the tubercular mischief.

We find that, during the later course of their illness, patients suffer from attacks of pyrexia very like true influenza. I have examined the sputum in many of these cases, during the past six years, and have found Pfeiffer's bacillus present on only two occasions. This differs from the observations of Williams,[2] of New York, who frequently finds Pfeiffer's bacillus in tuberculous sputum, long after an influenzal attack, and who even puts on tuberculous individuals the onus of spreading epidemics. The characteristic of the temperature reaction in tuberculosis is an exaggerated response to stimuli. The occurrence of a catarrhal "cold" in a tuberculous subject gives rise to pyrexia, and symptoms exceedingly like true influenza, but distinct bacteriologically. Such a catarrhal cold in a normal individual would produce a slight degree of pyrexia, but, in the more sensitive tuberculous subject, the rise is usually much greater. Daremberg[3] emphasises the importance of similar transient acute pyrexias in relation to gastric conditions, and holds the occurrence of such a pyrexia, with no sufficient apparent cause, to be diagnostic of tuberculosis. I have repeatedly seen such attacks diagnosed as "gastric influenza." In the above table, it will be noticed that a diagnosis of influenza was made in 51 per cent. of the cases, but, in none of these, so far as I could gather from the statements of patients, was the diagnosis confirmed bacteriologically. These attacks were identical with the pyrexias occurring later during treatment, in many instances in the same patient in which the influenza bacillus did not appear in the sputum. I venture to think that, had the diagnosis been confirmed by bacteriological examination, the anomalous nature of some of these attacks might have been detected, and perhaps a suspicion aroused as to the true nature of the condition, which would have saved the patient many months' stay in a sanatorium.

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[1] Burton Fanning: THE PRACTITIONER, March, 1902.

[2] Parks: *Bacteriology*, 1905, p. 324.

[3] Daremberg: *Tuberculose Pulmonaire*, 1905, p. 69.

It is hardly necessary to emphasise the frequency, with which hæmoptysis occurs with no physical signs. It is very frequently recurrent, and the diagnosis of tuberculosis is made at a later date, when physical signs, which are "only too obvious," have made their appearance. This is a matter of common experience among sanatorium physicians. Pleurisy has come to be regarded practically as a symptom of tubercle (when there is no other obvious cause), and such cases are now usually sent to a sanatorium for a course of open-air treatment with the most beneficial results. The physical signs may be slight or indefinite apart from the pleurisy, and, in many such cases, I have seen the diagnosis confirmed by the occasional appearance of the tubercle bacillus in the sputum. The result of coming to a "tentative diagnosis," so far as treatment is concerned, amply justifies itself; perfect health is restored after a relatively short period of residence, and the other signs in the lungs clear up rapidly. Similar good results follow the sanatorium treatment of recurrent influenza of a suspicious nature, and, when the condition turns out to be true influenza, the rest and bracing nature of the treatment rapidly restore the health. When, on examination, suspicious signs are found in the lungs, these almost invariably clear up rapidly.

It will be evident, on examination of the table, that many of these conditions must co-exist in individual cases. The most frequent symptoms, anorexia, dyspepsia, and constipation, are exceedingly common symptoms of non-tuberculous disease, but the point which I would emphasise, is that cases, showing these symptoms with suspicious associations, should be regarded as being pretuberculous. An association of languor with anorexia, more or less continued, and often without actual dyspepsia, but recurring in patients liable to influenzal attacks, or having an abnormal liability to colds, in short, a condition of "delicate health," is particularly suggestive of a chronic toxic poisoning. A soft, weak pulse, slow and regular in the recumbent posture, but becoming rapid on the least exertion, is very characteristic of the early stage of tuberculosis. This is associated with a cardiac condition demonstrable by the X-rays, which will be described later. Another suspicious symptom is perceptible dyspnœa on slight exertion with no cardiac lesion or anæmia; it is generally accompanied by some loss of weight and appetite.

The characteristic, in short, of the pretuberculous toxæmia is a muscular dystrophy affecting both voluntary and involuntary muscle, and manifesting itself in various ways. It accounts for the low weight from atrophy of the skeletal muscle, and induces the feeling of languor and unfitness for work, the feeling of "being easily tired" which is so frequently complained of. Atrophy of skeletal muscle is a sign of early as well as advanced disease. The same unfitness, as we have seen, affects the heart and also the intestinal muscle reducing peristalsis, and causing dyspepsia and constipation, though these are aggravated by a superinduced faulty chemistry and fermentation.

The bronchitis and "colds" of this stage are characteristic. A coryza starts in the nasal passages, and passes down, culminating in bronchitis. This may be localised from the first to one or other of the lungs, or it may be symmetrical, but, in clearing up, it usually leaves a localised patch of crepitation, which persists, or recurs from time to time. Seen in the early stage with the catarrhal condition active, it is almost necessarily diagnosed as bronchitis, but, seen later, it is called by various names: "catarrh of the lung," "catarrh of the apex," "weak lung," etc. Such conditions are really a catarrhal inflammation set up by the organisms responsible for the production of "colds" on a previously existing tuberculous patch.

The "inflammations," or pneumonias, of this stage have also distinguishing characteristics. A suspiciously large number give a history of "not clearing up properly," "no crisis," etc. The suspicious nature of such a condition is pointed out by Daremberg,[1] who holds that physical signs of pneumonic consolidation, causing less constitutional disturbance than lobar pneumonia, a persistent low pyrexia, no crisis, and no conditions indicating true broncho-pneumonia, are sufficient to justify a suspicion of latent tuberculosis.

[1] Daremberg: *Ibid.*, p. 56.

As I have already stated, these conditions may occur singly, but more often are associated, more or less persistently, with others on the list, and, with delicate health, they constitute the clinical picture of the pretuberculous stage. I would strongly urge the advisability, both for the sake of the individual, and as a preventive measure, of such cases being treated as suspected tuberculosis, because, in the instances where the principle has been recognised, the results are excellent. Provided the treatment is carried out where adequate precautions are taken against infection from cases of definite tuberculosis, the conditions are greatly benefited, even when, in the subsequent history of the case, there is no recurrence of suspicious symptoms or actual disease, and the tentative diagnosis is fortunately not confirmed.

RECENT METHODS OF DIAGNOSIS APPLICABLE TO SUCH CASES.

When once the suspicion has been aroused that a condition of ill-health is due to a toxic poisoning, possibly tuberculous in origin, we have various methods, clinical and scientific, which help to dispel or confirm our suspicions. Burton Fanning remarks that "it is some reflection on our methods that the majority of patients now presenting themselves for treatment have developed physical signs of disease which are only too obvious." Having regard to the fact that latent tuberculosis is so exceedingly common, and so apt to be lighted up by the many causes which predispose to the development of the affection, and also to the large number of cases of this chronic disease in its more advanced stage, which are at large among the community, the physician should always be on the alert to note suspicious signs, and should be ready to use all the means at his disposal for arriving at a correct diagnosis. Were all cases of pulmonary tuberculosis detected in the pretuberculous stage, no case-to-case infection could occur, and the extinction of the disease would be brought very perceptibly nearer.

TEMPERATURE.

It has been frequently pointed out that the temperature of a tuberculous patient is characterised, not necessarily by an elevation, but by poor regulation. It is well known that the rise of temperature, induced in the normal individual by exercise, rapidly subsides with rest, the normal being regained in less than half an hour. In the definitely tuberculous subject, the rise is more easily induced, and, the action of the heat-centres being sluggish, the rise is more pronounced and lasts longer. Daremberg[1] believes that a similar condition is characteristic of the pretuberculous stage before the onset of physical signs in the lungs. Burton Fanning,[2] on the other hand, is of opinion that the condition may be present in other states of chronic ill-health. Daremberg holds that a rise of temperature, persisting after exercise, is sufficient to confirm a diagnosis of tuberculosis in doubtful cases.

The test is one easily applied, but must be used with certain precautions. The temperature is best taken by the rectum, because the mouth temperature is practically under the control of the patient, and the slightest dyspnœa on exertion is apt to induce mouth breathing, which cools the mucous membrane. So much is this the case that two early observers insisted that mountain climbing lowered the body temperature. In order to make the test effectual, the temperature should be recorded after an hour's rest preceding the period of exercise, again after coming in, and a third time after an hour's rest in the recumbent posture. Should temperature perturbation be found to occur in a case from trivial causes, Daremberg recommends the application of the temperature test. I have known of the case of a lady, who was under my care, who suffered from pyrexia, sometimes as high as 101° F., after exertion or excitement, years before the tuberculous element became obvious. That such temperature conditions should exist during the pretuberculous stage is important confirmation of the hypothesis that, during the period of latent disease, there exists a toxæmia, long before the appearance of definite physical signs, and that such signs do not indicate the commencement of the disease.

X-RAY EXAMINATION OF THE CHEST.

Bouchard and Balthasar[3] have made careful measurements of the cardiac shadow with the planimeter, and find that there is a marked diminution in the size of the organ. The small size of the heart they believe to be characteristic only of the earliest stages, and they state that, in the later stages, the size is increased, thus differing from Lawson,[1] who states that enlargement is rare.[2] The diminution in the size of the heart was long ago pointed out by Fothergill,[3] who, with Bouchard and Balthasar, regards the small size of heart as being a predisposing cause of tuberculosis. It is difficult to see how a small size of heart could predispose, and I think it much more likely that the diminution is due to the pretuberculous condition, and is toxic in origin, being another instance of muscular dystrophy. A similar condition, I believe, affects the arteries, the muscular weakening being compensated by fibrous thickening, which is all the more striking, as the pulse associated with the thickening is almost invariably of low tension in uncomplicated cases.

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[1] Daremberg: *Ibid.*, p. 67.

[2] Burton Fanning: *Loc. cit.*, p. 33.

[3] Bouchard and Balthasar: *Trans. Tub. Cong.*, 1906, p. 550.

SKELETAL MUSCLE.

Daremberg[4] draws attention to a rather curious condition which he has observed in tuberculosis. He finds that, in cases with indefinite signs, which may or may not be tuberculous, there is atrophy of the limbs on the side of the lesion in cases due to tubercle. The skeletal muscle is irritable and gives degrees of contraction on tapping, but this is not diagnostic, being found in other conditions of malnutrition.

THE ULNAR REFLEX.[5]

This is a skin reflex present in practically all cases of advanced tuberculosis, and rarely present in other conditions of intoxication, such as cancer, etc., and not present in normal individuals. It is elicited by scratching the skin of the anterior surface of the forearm on the ulnar side with a pointed instrument, which leads to a contraction of the abductor minimi digiti. In some cases of unilateral lung involvement this reflex is present only on the affected side.

In other cases, and this is more frequent, it is present on both sides, even in early cases.

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[1] Lawson: "X-Rays in Diagnosis," THE PRACTITIONER, Extra Number.

[2] Dr. Riddell of the Glasgow Royal Infirmary tells me that in his cases he does not uniformly find a diminution in size in tuberculosis.

[3] Woods Hutchinson: *Comp. Path.*, p. 299.

[4] Daremberg: *Loc. cit.*, p. 75.

[5] *Phipp's Institute 1st Annual Report.*

While this reflex is not pathognomonic of pulmonary tuberculosis, its frequent occurrence in this disease and its rarity in other conditions are suggestive. Its presence in the routine examination in dispensary work has led, on several occasions, to the examination of the chest and the discovery of an unsuspected tuberculosis.

The arm of the patient should be flexed and held loose. The operator grasping the fingers loosely so as to be sure of relaxation of the fingers, the point of a pin is rapidly drawn with moderate force along the ulnar side of the anterior surface of the arm from elbow to wrist. A distinct wrinkling of the external aspect of the hypothenar eminence is produced.

[1] "The ulnar reflex was present in 14 out of 15 advanced cases, and according to Dr. Cameron's records, in 71 of the 173 dispensary cases examined. A careful study of the control cases during the year has shown that this reflex is of rare occurrence outside of pulmonary tuberculosis."

THORACIC PERIMETER.

The most recent observations on the thoracic perimeter are those of Joffres and Maurel,[2] who have come to the following conclusions: —That the thoracic perimeter is diminished in tuberculosis, and the normal ratio of 8 c.m. per kilo. body weight is always diminished. If the ratio is diminished in cases with wasting, it would be of interest to know whether the same holds in pretuberculous cases before any marked degree of emaciation has been established. Boureille[3] has made measurements of the respiratory activity in relation to the pretuberculous stage. Ninety-six cases, with doubtful signs and inconclusive symptoms, were compared with 385 healthy individuals. In the healthy chests, the expansion was found to vary between 6 and 14 c.m. as the extreme limits, while 95 per cent. of the cases suspected of tuberculosis were below 4 c.m., and the greater majority of these between 2 and 3 c.m. "These measurements," he observes, "rarely deceived me in cases with no physical signs and where sputum was absent."

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[1] Extract, *Phipp's Institute 2nd Annual Report*, p. 134.

[2] Joffres and Maurel: *Trans. Tub. Cong.*, 1906, p. 569.

[3] Boureille: *Trans. Tub. Cong.*, 1906, p. 570.

EXAMINATION OF THE SPUTUM.

The presence of tubercle bacilli in the sputum of course at once clinches the diagnosis, but their absence, even during months with frequent examinations, does not exclude the possibility of tuberculosis.

EXAMINATION OF THE URINE.

The excretion of chlorides and their degree of concentration in the blood seems to have some unexplained relationship to pulmonary tuberculosis. Richet found that the giving of salt with raw meat hindered its curative action. Linoissier has also shown that chloride of sodium delays peptic digestion of proteid. Daremberg has frequently improved the digestive conditions in dyspeptic cases by diminishing the intake of salt. Daremberg[1] believes that diminished chloride excretion is of diagnostic value in all doubtful cases. Piery and Deney[2] find, however, that chloride excretion varies with the type of the disease, and, while their results coincide with those of Daremberg in certain types of tuberculous lung mischief, they are in many respects contradictory. Possibly a faulty control of chloride intake may account for the discrepancy. The diagnostic value of chloride estimation must, therefore, be considered as *sub judice* pending more accurate and complete observations.

TUBERCULIN TEST.

The injection of large doses of tuberculin, with the object of inducing a reaction in suspected cases, has been much lauded, but, so far, has not been largely employed by practitioners. It seems as if the light thrown on the action of tuberculin by the opsonic index would further discourage its use, from the possibility of inducing an undesirably prolonged negative phase, with a dose insufficient to produce temperature reaction, and the consequent possibility of inducing increased activity in a latent focus.

BLOOD EXAMINATION.

A normal or slightly increased number of red cells with a diminished hæmoglobin percentage is usually found. The differential count in early cases of advancing disease shows a low percentage of mononuclears, this percentage rising in cases undergoing improvement.[1] I have noted that after tuberculin injections, sufficient to cause a temperature rise, there occurs a diminution of the white cells coincident with the rise in temperature, this decrease being mononuclear.

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[1] Daremberg: *Loc. cit.*, p. 73.

[2] Piery and Deney: *Trans. Tub. Cong.*, 1906, p. 410.

OPSONIC INDEX.

We should expect to find a low index from what we know of the behaviour of the index in the later stages of the disease. I have estimated the index in a considerable number of doubtful cases sent for diagnosis, and have always found a low index, usually about ·7–·8.[2] It is well, however, to note that the presence of a pathological relationship between any organism and specimen of serum may manifest itself either by a raising or lowering of the index. This means of diagnosis promises to prove of great value.

In conclusion I would recapitulate the points which I have endeavoured to emphasise.

I. The early physical signs of pulmonary tuberculosis do not correspond with the commencement of the disease.

II. For a prolonged period, previous to diagnosis, the disease is advancing from a small deep-seated lesion and causing a toxæmic condition setting up various symptoms, and modifying the course of intercurrent diseases.

III. The variety and continuance of these symptoms constitute a clinical picture, which, when recognised by experience, should suggest the possibility of latent tuberculosis.

IV. Various methods, clinical and scientific, are available for rendering the diagnosis more certain.

V. In cases, in which a tentative diagnosis is followed by appropriate treatment, the results justify the course adopted.

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[1] Simon and Spillman: *Trans. Tub. Cong.*, 1906, p. 432.

[2] Dr. Elizabeth Fraser in the *Glasgow Medical Journal*, April 1907, recommends as an absolutely accurate test for suspected cases, whose index falls within the normal limits ·8-1·2, the injection of $\frac{1}{1000}$ m g. of T.R., which causes a negative phase in tuberculosis but none in the healthy individual.

THE EARLY DIAGNOSIS AND CURE OF THE PRESUPPURATIVE STAGE OF AMŒBIC HEPATITIS.*

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No one, who has had a large experience of the early stages of amœbic abscess of the liver in a tropical climate, can fail to have been struck by the great frequency with which definite clinical symptoms of suppuration in this organ are preceded by weeks or even months of fever, during which there may be few or no symptoms pointing to the liver as the cause of the trouble, and which is commonly ascribed to and treated as "malaria." Moreover, even when marked symptoms of acute hepatitis are present, it is frequently impossible to decide whether an abscess has already formed or no, without resort to an exploratory operation, which only too often gives a negative result in cases in which an abscess of the liver is found at a later date.

In a paper,[1] published a year and a half ago, I discussed the value of leucocytosis in acute hepatitis, and concluded that its presence in a marked degree was generally an indication that suppuration had already taken place, but in the slighter degrees it may be present in acute hepatitis without actual suppuration, and that this early stage of amœbic hepatitis may sometimes be cured, and suppuration prevented, by the administration of large doses of ipecacuanha. Since then I have repeatedly confirmed this observation, and can now go a step further in support of that position.

In the course of a comprehensive investigation of 13,500 cases of fever, I have met with a most interesting group, which appear to me to throw much light on the early presuppurative stages of amœbic hepatitis, and to indicate that this disease may frequently be recognised by the blood changes, when in a stage which admits of rapid cure, and so prevented from drifting on into the much more serious suppurative stage, as is now so commonly their fate. Further, these cases constitute

* Read before the Royal Medical and Chirurgical Society.

a definite class of fever, usually of a chronic intermittent type sometimes with no very definite symptoms of hepatitis, and rarely with any dysentery. They may be recognised, or at least strongly suspected, by the presence of a moderate degree of leucocytosis, generally of the type which[1] is common in amœbic abscess of the liver, namely, one in which the proportion of polynuclears is either normal, or only slightly in excess. Further, this kind of fever rapidly yields to large doses of ipecacuanha, in the absence of symptoms of dysentery or even of hepatitis, and the formation of tropical abscess of the liver is thus prevented. If this proposition can be substantiated, a great advance will be made in the prevention of one of the most dangerous diseases of certain tropical countries.

For convenience of reference, all the cases of non-suppurative hepatitis, met with in the Calcutta European Hospital fever series, during the year subsequent to those dealt with in the paper, already referred to, on blood counts in amœbic hepatitis, have been embodied in the accompanying table, and classed in accordance with whether they showed clinical symptoms of dysentery and hepatitis, hepatitis alone, or no definite signs of either at the time the blood examination was made. The patients were under the care of Drs. B. Chatterton, J. C. H. Leicester, and J. G. Murray, all of the Indian Medical Service, to whom I am greatly indebted for permission to examine the blood, and make use of the hospital notes.

I.—CASES OF ACUTE HEPATITIS COMPLICATED WITH DYSENTERY.

Beginning with the simplest and most straightforward cases, we first have Nos. 1 to 3, in which an acute hepatitis followed symptoms of dysentery, leucocytosis being present in each, while, as the liver was also enlarged and very tender, liver abscess was suspected. In No. 3 exploratory puncture of the liver in six places was done with a negative result. In all three cases, the ipecacuanha treatment was followed by cessation of the fever, and other acute symptoms, within from 2 to 4 days, although they had been present for 15, 34, and 41 days respectively before this drug was given in large doses. The following are the principal points of interest in these

TABLE OF BLOOD COUNTS AND IPECACUANHA TREATMENT OF PRESUPPURATIVE AMŒBIC HEPATITIS.

Number.	Sex.	Age.	Bowels.	Liver.		Days' Fever before Ipecacuanha Treatment.	Days' Fever after Ipecacuanha Treatment.	Red Corpuscles.	White Corpuscles.	Ratio of White to Red.	Polynuclears.	Lymphocytes.	Large Mononuclears.	Eosinophiles.	Remarks.
				Inches below Costal Margin.											
I. Cases with Dysentery and Hepatitis treated with Ipecacuanha.															
1	M	38	Dysentery	1	Tender -	41	2	3,600,000	15,250	1-236	83	13	4		
2	M	31	Do. -	2½	Do. -	15	4	—	Leucocytosis	—	78	16	2	4	
3	M	33	Do. -	1¼	Do. -	34	3		Do.	—	79	12	6	3	Aspirated.
II. Cases of Hepatitis without Dysentery treated without Ipecacuanha.															
4	M	35	Normal -	½	Tender -	49	—	3,000,000	24,000	1-129	78	15	7	0	Aspirated.
5	M	45	Do. -	2	Do. -	39	—	3,610,000	28,500	1-126	76	21	3	0	Aspirated.
6	M	34	Do. -	¼	Do. -	34	—	3,910,000	9,500	1-416	78	8	3	2	
III.—Cases of Hepatitis only without Dysentery treated with Ipecacuanha.															
7	M	40	Irregular -	1½	Tender -	15	1	4,820,000	21,000	1-230	78	15	5	2	
8	M	38	Normal -	1	Do. -	14	4	4,600,000	17,750	1-233	87	7	5	1	
9	M	31	Diarrhœa -	0	Do. -	25	5	—	Leucocytosis		80	15	4	1	
10	M	35	Constipated	2½	Do. -	50	5	5,380,000	17,000	1-316	76	16	6	2	Aspirated.
11	M	38	Normal -	0	Do. -	13	6	4,910,000	9,500	1-517	79	16	4	1	
IV.—Cases without Dysentery or Liver Symptoms treated with Ipecacuanha															
12	M	40	Normal -	1	Normal -	6	—	4,290,000	15,250	1-272	81	11	7	1	Alcoholism.
13	M	28	Do. -	¼	Do. -	53	15		Leucocytosis	—	—	—	—	—	Aspirated.
14	M	24	Irregular -	1	Do. -	35	2	4,660,000	20,750	1-225	—	—	—	—	
15	M	32	Constipated	0	Do. -	45	12	3,340,000	17,250	1-193	74	22	3	1	

cases, the temperature charts of which I show you on the screen :—

Case 1.—A male, aged 38, who had been in hospital two and a half months previously for a slight attack of hepatitis, which yielded to ammonium chloride treatment in three days. He had suffered from dysentery on and off for seven months before this first attack of hepatitis, and, on readmission, was passing mucus and blood, but showed no signs of hepatitis. The dysentery improved under bismuth, but the irregular intermittent fever continued in spite of quinine, and, one month after he came into hospital, I found slight increase of the leucocytes, but not amounting to an actual leucocytosis. Six days later, his liver having become tender, a second examination showed 15,250 white corpuscles per cubic millimetre, a leucocytosis having now developed. Although all active symptoms of dysentery had long ceased, I suspected a latent form of the disease as the cause of the hepatitis, and advised the ipecacuanha treatment, which was followed within two days by a permanent cessation of the fever, although the case was just one of those which ordinarily drift on into liver abscess.

Case 2.—This man was also admitted for dysentery, together with signs of acute hepatitis and a leucocytosis, but the remittent temperature fell to normal in four days under ipecacuanha, although there was a slight relapse of the dysenteric symptoms four days later, but at the end of 16 days the fever finally left him. A Widal test with Shiga's bacillus gave a negative result in a dilution of 1 in 20, so the dysentery was probably amœbic in nature. This case was a straightforward one of dysentery followed by hepatitis, yielding to the well-known ipecacuanha treatment as advised, when dysentery is present, by Sir Patrick Manson and other authorities.

Case 3.—A male, aged 33, whose illness began three weeks before admission with dysentery, lasting for two weeks. He was passing loose greenish stools, while his liver was enlarged and tender, and leucocytosis was present. X-rays showed loss of movement of the diaphragm on the right side, and, liver abscess being suspected, the organ was punctured in six places under chloroform, with a negative result. He had been given ipecacuanha in two-grain doses, with only some lessening of the pyrexia resulting, but two weeks after admission he was given 20-grain doses twice a day, and on the third day his temperature finally fell to normal, and he rapidly recovered (Chart I.).

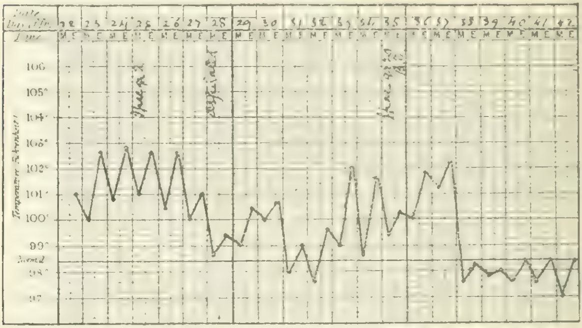

Chart I. (*Case* 3).

Remarks on Group I.—These three cases illustrate the well-known treatment of cases of dysentery, complicated by acute hepatitis, by large doses of ipecacuanha, although the rapidity with which the hepatitis, with a definite leucocytosis, yielded to the drug, in spite of the dysenteric symptoms being in abeyance in two of them, is noteworthy, for it is just such cases which so commonly drift on into the suppurative stage of the disease if this treatment is neglected, as it too often is in the tropics at the present day.

II.—CASES OF HEPATITIS WITHOUT DYSENTERY NOT TREATED WITH IPECACUANHA.

Next I come to cases of acute hepatitis without any recent dysentery, which I have divided up in accordance with whether they were treated with large doses of ipecacuanha or no, and will first deal with the three cases not treated with this drug (Nos. 4 to 6).

Case 4.—A seaman, aged 38, admitted for acute hepatitis with a leucocytosis but no dysentery. X-rays showed reduced movement of the diaphragm on the right side, so, eight days after admission, the liver was aspirated in two places, only blood being obtained, so the abdomen was opened, the organ palpated and aspirated in three more places, also with a negative result. Irregular intermittent fever continued for 12 days after the operation, and, a few days after the pyrexia ceased, the leucocytosis was also found to have disappeared. He continued to get occasional slight rises of temperature for another month, but eventually left hospital apparently well, after being for 62 days under treatment, and, as he went to England, I was unable to follow up his case further, so cannot say if he eventually developed a liver abscess or not. He was not treated with large doses of ipecacuanha while in hospital, and his recovery was a very protracted one.

Case 5.—A male, aged 45, admitted for low intermittent fever and hepatitis, his blood showing a very marked leucocytosis, 28,500 per cubic millimetre, and whose liver was also punctured in several places without any pus being found. The pyrexia ceased after 39 days, and he left hospital, at his own request, much improved, after 57 days' stay. He was re-admitted for another much slighter attack of hepatitis 10 months later.

Case 6.—Admitted for mild hepatitis, with low intermittent fever up to about 100 only, which lasted for 34 days under quinine treatment without any very acute symptoms appearing. There was no leucocytosis in this case, which was probably alcoholic in origin, but is included, as it is one of a consecutive series of unselected cases.

Remarks on Group II.—The first two of this group are typical of the class of acute hepatitis without dysentery, in which leucocytosis, pointing to liver abscess, is present, and exploratory operation becomes necessary in order to decide whether a liver abscess has formed or no. A negative result being not uncommonly followed by a slow improvement and ultimate recovery, although they are apt to relapse, and an abscess is frequently found at a later date. They will serve for comparison with the next group of similar cases, in which the ipecacuanha treatment was carried out.

III.—CASES OF HEPATITIS WITHOUT DYSENTERY TREATED WITH IPECACUANHA.

Cases 7 to 11 all presented signs of acute hepatitis without any recent dysentery, four out of the five also showing marked leucocytosis. They were all treated with ipecacuanha.

Case 7.—Had been in hospital for a short attack of hepatitis four months before. No history of dysentery, but had suffered occasionally from a watery diarrhœa, alternating with constipation—a not infrequent occurrence in amœbic hepatitis. He suffered for 15 days from fever of a low remittent and intermittent form, at the end of which he was put on the ipecacuanha treatment, and two days later his temperature finally fell to normal, and he made a good recovery, returning to work after 32 days in hospital. However, he returned five months later with a liver abscess, which was cured by aspiration and injection of quinine into the cavity, as already reported.[2] After which he went to work again, and remained quite free from fever for eight weeks, when he returned with a second abscess in a different part of the liver to the one which had been injected with the quinine. A similar treatment was tried on the fresh abscess, but this time it failed, and eventually it had to be opened and drained, since which he has had no return of his trouble.

In connection with this method of treating early cases of liver abscess by aspiration and injection of soluble bihydrochloride of quinine into the cavity to kill the amœba, when it is found to be free from bacteria, I regret to have to report that in three further cases, treated by Captain J. G. Murray, I.M.S., at the General Hospital, Calcutta, it failed, and the open operation had to be resorted to, and I am informed that in Bombay a similar experience was obtained in two cases, so that it has not fulfilled my anticipation that it would succeed in most of the cases of tropical abscess. On the other hand, I have been informed of several cases in which it has proved successful in other hands than my own, so, in view of the rapid and comparatively painless cures it sometimes effects, and its harmlessness, in our Calcutta experience in those cases which have eventually to be cut open, I still think it worthy of trial in early deep-seated amœbic abscesses of the liver.

Case 8.—A male, aged 38, admitted for acute hepatitis without any history of dysentery and no present bowel trouble, his temperature showed the rapid oscillations produced by the frequent profuse perspirations so common in acute liver inflammation, a marked leucocytosis was present, and he had occasional rigors. Nevertheless, the pyrexia and all the acute symptoms yielded in four days to 20-grain doses of ipecacuanha morning and evening (Chart II.).

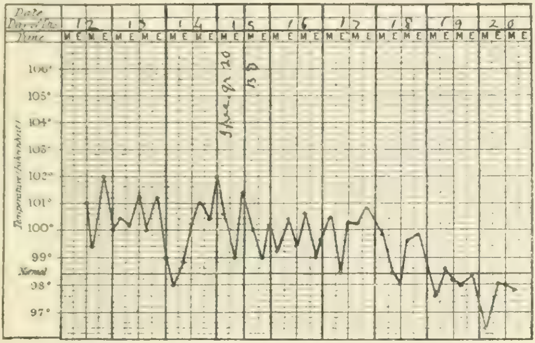

Chart II. (*Case* 8).

Case 9.—A male, aged 31, admitted with a sub-acute hepatitis with low intermittent fever, watery diarrhœa, and a slight degree of leucocytosis, all the symptoms disappearing after five days of the beginning of the ipecacuanha treatment.

Case 10.—A male, aged 35, admitted for acute hepatitis with constipation, and low intermittent fever, which improved for a time under quinine, only to return shortly after. A month after his admission I was asked to examine the blood and found 17,750 leucocytes per cubic millimetre, so the liver was punctured with a negative result. He was then put on 5 grains of ipecacuanha three times a day, and four days later was injected with antistreptococcus serum, the temperature finally falling the next day, and he made a good recovery. In this case it is difficult to say whether the happy result was due to the ipecacuanha or to the serum, but I am inclined to think it was the former, because two and a half months later he returned with an amœbic abscess of the liver, which was opened and drained with an ultimately fatal result ; so that his earlier attack was pretty certainly also amœbic in origin, and so likely to have yielded for a time to the small doses of ipecacuanha given.

Case 11.—Male, aged 38, who had suffered from dysentery 12 years before, bowels now normal, but symptoms of acute hepatitis without leucocytosis and low remittent fever, which declined when the ipecacuanha treatment was commenced, and finally ceased after six days, the case being a somewhat mild one (Chart III.).

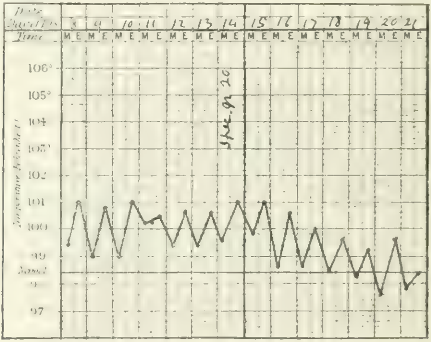

Chart III. (*Case* 11).

Remarks on Group III. These cases of hepatitis without dysentery are precisely similar to those of Group II., but the pyrexia and other symptoms rapidly ceased under the ipecacuanha treatment within from 1 to 6 days, although they had previously been present for from 13 to 37 days, and in No. 10 aspiration had been done on account of a suspicion of a liver abscess being present. In short, the effects of this treatment were precisely the same as in the first three cases in which symptoms of dysentery as well as hepatitis had been evident.

IV.—CASES WITHOUT DYSENTERY OR LIVER SYMPTOMS TREATED WITH IPECACUANHA.

Cases 12 to 15 all showed leucocytosis without symptoms of either dysentery or acute hepatitis being present, fever, for which no definite cause could be found, being the condition for which they were admitted. As there was no obvious cause for the leucocytosis, I suspected that latent amœbic dysentery and the insidious form of hepatitis, which sometimes precede actual abscess formation, might be present, and, therefore, suggested a trial of the ipecacuanha treatment, which had proved so efficient in the more acute forms of amœbic hepatitis. Although there was neither pain nor tenderness of this organ present when they were first admitted, in spite of slight enlargement of the liver in three of the four, the results were most satisfactory.

Case 12.—A seaman, aged 40, with alcoholic history, whose pyrexia ceased after 6 days without ipecacuanha, but who was subsequently treated with it on account of the presence of leucocytosis, in order to try to prevent a recurrence of the liver trouble, as the fall of temperature might have been temporary improvement only. This case must be acknowledged to be a doubtful one.

Case 13.—Male, aged 28, admitted for fever of 3 weeks' duration, which persisted under quinine treatment, although no cause could be found for it. Bowels normal, both sides of

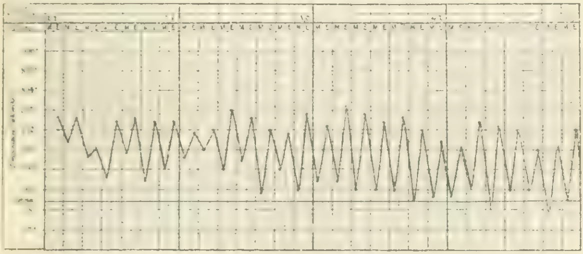

Chart IV. (*Case* 13).

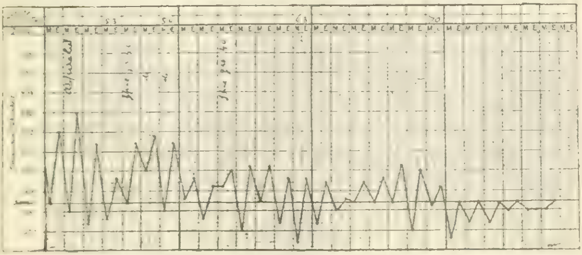

Chart IV. (*Case* 13—*continued*).

the diaphragm moving well as seen by the X-rays, liver slightly enlarged, but not tender, although becoming larger. Leucocytosis was found by me both 1 week and 3 weeks after admission, so the liver was explored for abscess with a negative result. Three days later, or 53 days after the fever commenced, the ipecacuanha treatment was commenced, and on the fourth day the temperature was normal, but slight low fever up to 100° F. recurred for several days more, after which convalescence set in as shown in Chart IV.

This was a most remarkable case as the nature and cause of the fever was quite a puzzle until the presence of leucocytosis led to a suspicion that latent amœbic dysentery and hepatitis might be at the bottom of it, while the rapid success of the ipecacuanha treatment in such a persistent fever appears to me to lend some support to the correctness of this view of the case.

Case 14.—Male, aged 24, admitted for irregular intermittent fever, not yielding to quinine, and with no obvious cause. As over 20,750 leucocytes were found, the ipecacuanha treatment was again adopted with the happiest results, the fever finally ceasing 2 days later, and convalescence being quickly established as in the preceding case (Chart V.).

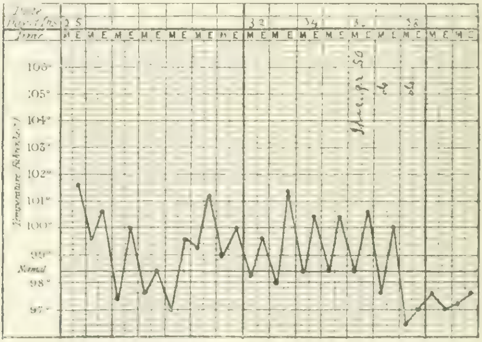

Chart V. (*Case* 14).

Case 15.—Male, aged 32, admitted for fever with no ascertainable cause, and resistent to quinine. Bowels constipated, diaphragm moving well. A well-marked leucocytosis was found, 40 grains of ipecacuanha were accordingly given on 2 consecutive days followed by 2 grains twice a day, but with apparently very little effect on the fever. Hypodermic quinine was then tried without result, and, on the 16th day after admission, 30 grains of ipecacuanha were again administered, and repeated in doses diminished by 5 grains each day, a treatment which was followed by a final cessation of the pyrexia in 2 days. In this case, the fever had lasted for 45 days before the first trial of ipecacuanha, so that it is, perhaps, not surprising that two large doses failed to stop it, and it had to be repeated again a few days later. The ultimate result was as satisfactory as in the previous cases, so that I do not think that the repeated apparently good results of this treatment can be simply a coincidence.

Remarks on Group IV.—The remarkable feature of these last four cases is that there were no bowel or liver symptoms to make one suspect the presence of amœbic hepatitis, until an otherwise unexplainable leucocytosis was found on examining the blood. Yet on administering large doses of ipecacuanha in three of the cases after the fever had resisted quinine and other treatment for 35, 45, and 53 days respectively, the pyrexia and other symptoms subsided in much the same way as in the previous groups, showing definite symptoms of hepatitis with or without dysentery. Moreover, judging from a prolonged experience of similar cases before this treatment was so commonly used in the Calcutta European Hospital, patients of this class do not rarely drift on into liver abscess formation if their true nature is not discovered, and the ipecacuanha method vigorously used, while I know of nothing but the presence of leucocytosis which will allow of their nature being suspected in this early stage, when they are so readily amenable to medical treatment. I may add that several very similar cases seen in consultation have furnished equally satisfactory results to those dealt with above under ipecacuanha.

THE TYPE OF THE LEUCOCYTOSIS IN AMŒBIC HEPATITIS.

In the paper already referred to, I pointed out that the leucocytosis in amœbic abscess is somewhat peculiar, in that the proportion of the polynuclears is commonly comparatively little, if at all, increased, as it is in ordinary suppurative conditions. A reference to the table will show that a similar type is present, as a general rule, in the presuppurative stages of amœbic hepatitis, which is a strong point in favour of the view that these cases are also secondary to an amœbic dysentery, usually of a latent nature. Thus, out of 13 cases, in which the differential leucocyte count was done, in none were as many as 90 per cent. of polynuclears present, while in only three were over 80 per cent. found. I have several times found this type a point of diagnostic value in favour of this affection rather than some other suppurative or acute inflammatory condition.

THE IMPORTANCE OF ALWAYS TRYING THE IPECACUANHA TREATMENT BEFORE OPERATING.

Another striking point in this series is the fact that, in no less than one-third of them, an unsuccessful exploratory operation was undertaken before the disease was rapidly cured by the ipecacuanha treatment, this having been done in several of them, at my own suggestion, before I had fully realised how often a marked leucocytosis is present in the readily curable early presuppurative stage of this amœbic hepatitis. In view of the results of that treatment now brought forward, I am strongly of the opinion that all cases of this disease should be treated with large and repeated doses of ipecacuanha, whenever there is any doubt remaining as to whether suppuration has already taken place or not, before any exploration of the liver is carried out, for this rule will certainly prevent some unnecessary surgical measures. Moreover, repeated puncturing of the liver with large aspirating cannulas is by no means without danger, as shown by the number of recorded cases of fatal hæmorrhage following them, while it is especially, in these cases of very acute congestion of the organ without actual suppuration, that such deplorable deaths have occurred.

THE PATHOLOGY OF THE PRESUPPURATIVE STAGE OF AMŒBIC HEPATITIS.

The explanation of the series of cases just described remains to be considered.

My very favourable position for the study of liver abscess in the tropics, enabled me to record, in 1902,[3] that I had found living amœbæ in 75 consecutive cases of liver abscess, in which scrapings of the wall were examined within less than twelve days of the cavity being opened, while the great majority of them were otherwise sterile when first incised. Further, I showed that, when both the clinical history and post-mortem records of cases were available, dysentery had been noted in 90 per cent. of the cases, always, in my experience, of the amœbic type. In 18 per cent. of the cases, the clinical history of dysentery was negative, but nevertheless dysenteric ulcers were found in the large bowel after death, the disease having been present in a latent form: a sequence of events which has since then repeatedly come within my knowledge. I, therefore, hold that the amœba dysenterica is constantly present in early amœbic abscess of the liver, although it may very occasionally die out in chronic cases, and that the liver affection is secondary to amœbic dysentery, although this may frequently be present in a latent form, and give rise to no typical clinical symptoms, owing to its ulcers being limited to the cæcum and ascending colon.[4]

Now if the above statements are well founded, then it follows that, in the presuppurative stages of amœbic hepatitis, in the absence of any clinical history or actual systems of dysentery, there must be a latent form of that disease present giving rise to irritation of the liver on account of these protozoal organisms reaching the organs by the portal circulation, and, if this source of irritation can be removed, the hepatitis should subside. Now it is in just those tropical climates, where liver abscess and amœbic dysentery occur, that ipecacuanha is looked on as a specific in many cases of dysentery, while I have been informed by several medical men, with experience of dysentery in countries where amœbic abscess of the liver is not seen, that this drug is useless in the dysenteries of bacterial origin which they there had to deal with. Personally, I look on ipecacuanha as invaluable in the treatment of amœbic dysentery, in fact as a specific against that disease, and in Lower Bengal, where amœbic liver abscess is so common, I regard this drug as second only in importance to quinine itself. If this is so, it is easy to understand how large doses of ipecacuanha (not less than 20 to 40 grains once or twice a day some twenty minutes after a dose of tincture of opium) may rapidly abort an early presuppurative amœbic hepatitis by curing the latent amœbic dysentery that produces it, although I find no recommendation of the drug in some of the standard works on tropical medicine in acute hepatitis, except when symptoms of dysentery are actually present. The cases narrated are, I venture to think, sufficiently striking to warrant the general extension of the ipecacuanha treatment as a routine method in cases of hepatitis, in countries where amœbic abscess of the liver occurs, in the absence of actual history or symptoms of dysentery, and that this treatment should always be given a trial before operative measures are undertaken in all cases, in which any doubt exists as to whether or no actual suppuration has yet occurred.

Lastly, we also know that amœbic abscess of the liver may arise sometimes in a most insidious manner, there being no very definite indication of hepatitis for a long time in spite of persistent fever. In such cases, we may also have a latent amœbic dysentery as the exciting cause of the chronic fever, so that it is but one step further to treat these also with large doses of ipecacuanha as soon as the nature of the case can be determined, while I have shown that they may often at least be suspected from the presence of the type of leucocytosis already mentioned. The rapid cessation of the previously persistent intermittent fever without symptoms of hepatitis under such treatment, in some of the cases narrated, is the best evidence of the correctness of this argument. I am sanguine enough to hope that, when these methods of early diagnosis, and prompt ipecacuanha treatment of the presuppurative stages of amœbic hepatitis become generally known and practised, much will be done to lessen the occurrence of tropical abscess of the liver, with its very high mortality and prolonged suffering especially in the case of Europeans, who come early under observation for the fever which so constantly precedes, often for several weeks, actual breaking down of the liver substance.

REFERENCES.

[1] *B.M.J.*, November 11, 1905.
[2] *B.M.J.*, June 16, 1906.
[3] *B.M.J.*, September 20, 1902.
[4] *B.M.J.*, June 6, 1903.

POST-PARTUM HÆMORRHAGE.

By E. STANMORE BISHOP, F.R.C.S. (Eng.),
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In The Practitioner for April, 1907, there is an article by Dr. G. Ernest Herman of the London Hospital on this subject which deserves notice, partly because of the well-known position and reputation of the writer, and partly because it marks, I conceive, a distinct stage of advance in the discussion. It shows unmistakably a wish to recede from the position hitherto taken up, and to disavow, so far as possible, the teaching which has so long been current. Dr. Herman even goes so far as to express ignorance of the existence of any such teaching. He does not know, he says, where such advice as "that in order to check post-partum hæmorrhage, the uterus must either be made to contract or some astringent preparation such as perchloride of iron must be applied to its interior," is given. In the light of what follows it is difficult to believe that he is serious. In case there are any others who are equally in the dark, for his and their benefit, I beg to refer him to the following works, and to the actual pages, in which he will find that teaching plainly set forth, without the least ambiguity:—

Playfair, *Science and Practice of Midwifery*; 9th edition, 1898, Vol. 2, p. 123, *et seq.*; Galabin, *Manual of Midwifery*, 6th edition, 1904, pp. 802, 803, 805; Eden, *Handbook of Midwifery*, 1st edition, 1906, pp. 344 and 349: in this work adrenalin is preferred to iron, but the idea is the same; Dakin, *Handbook of Midwifery*, 1st edition, 1897, pp. 480 and 483.

There are others, whose names can be given, if Dr. Herman wishes for further confirmation; amongst them is one, whom it would not be fitting to pass with a mere reference; an actual quotation is necessary; that author is Dr. Herman himself. In his most valuable work on *Difficult Labour*, p. 333, is the following paragraph:—

"The modes of stopping bleeding after labour may be divided into three groups according to their principal aim, which is:—1. To make the uterus contract; 2. To clot the blood; 3. To compress the bleeding veins." And then he proceeds to describe the details of what I have called "experiments," but which he prefers to denominate "manipulations."

In THE PRACTITIONER article, Dr. Herman speaks of the injection of perchloride of iron as not merely dead, but "extinct." His actual words are, "I know not where this teaching is given. I thought the treatment by the injection of perchloride of iron was now extinct." He will not, I am sure, complain if we turn to his book, *Difficult Labour*, again in order to see what he has said about this measure there. It is evident that he includes it in his second class of remedies for post-partum hæmorrhage, indeed, p. 336, he distinctly says so. Further, he goes on to say, "The injection of perchloride of iron solution (1 to 6) stimulates the uterus to contract and clots the blood. It is the clotting that is valuable, for the stimulant effect can be got by other means. The cases, in which the clotting is required, are those in which the uterus has lost its contractile power" (note the admission that such is the case), "therefore you leave off trying to make it contract, and act directly on the blood." When I read this in the revised edition of his work published in 1900, I naturally thought that this was simply his view in that year; and that, in his later edition, published last year, I should find a totally altered text, more in consonance with his present declaration that such teaching is now extinct, so I sent for the 1906 edition and found . . . precisely the same words.

It is perfectly true that, farther on, he enumerates the dangers of this practice, and gives the preference to bimanual compression of the uterus, but does he imagine that a practitioner, face to face with the imminent death of his patient, having tried drugs—and failed; having tried packing—and failed; having tried injection of hot water—and failed; having tried bimanual compression—and failed, will be deterred by any remembrance of an academic discussion of its dangers, but will snatch at the chance of success of a measure thus brought to his notice, the actual proportions of the preparation being given, and the reason for its employment explained, and try this "manipulation" also—and fail.

In Fig. 125, p. 341, in *Difficult Labour,* Dr. Herman delineates a uterus treated by his method of bimanual compression. I imagine that Dr. Herman has not included it as a real representation of what a uterus in danger of serious post-partum hæmorrhage is like, but as showing, to the utmost advantage, his plan of bimanual compression ; it is, however, calculated to give a student -and this Manual is, I suppose, written for students and young practitioners—an altogether inaccurate idea of the actual state of affairs. The uterus which is the subject of dangerous post-partum hæmorrhage is not a thick-walled, easily-defined object such as is there shown. It is a thin, soft, limp, widely-spread organ, very difficult to differentiate from the parts around. It is so limp and large that it cannot be entirely grasped by the hands of the obstetrician ; parts of it may be grasped, but others elude his grip and escape all compression. If I am wrong in my surmise, Dr. Herman and I are evidently thinking and talking about two entirely different things, and it is no wonder that we disagree as to the manner of treating them.

And this probably explains also his reluctance to abandon frankly his idea of compressing the individual bleeding points. He speaks as if it were possible to make out easily the location of the bleeding placental site. As everyone knows, this may be anywhere on the internal surface ; in such a large, limp, soft-walled cavity, the pressure of the fist, applied internally, and reinforced without by the pressure of the other hand, may easily be exerted on a totally non-offending area of its circumference, whilst the really important patch of bleeding mucosa may be far away up as high as the stomach, or under the liver, entirely uncompressed, entirely unaffected.

This is apparently one of the most difficult obstacles arising in men's minds which anyone, who holds the views I do, has to overcome ; otherwise so many writers would not express a preference for internal interference. The objections to such interference being so many and so obvious, I presume that it satisfies one's natural desire to do something daring, to show that one has not been deterred by any difficulty, any risk in one's endeavours to save life ; but it is well to remember that the risk in these cases is run not by us but by the patient ; that, if by good luck we may be successful, the patient, and not we, may have to pay for her life by a long period of more or less acute suffering from metritis, salpingitis, and pelvic peritonitis ; conditions which every gynæcologist has been able to trace to too energetic manual interference on the part of the attendant at precisely such times as these. Calm reflection will, however, show that what may possibly be obtained in this way can, without risk to the patient, be obtained far more certainly by compression of the one tube, which supplies all the important bleeding arteries; that it is far more certain to succeed, as well as more easy to compress a tube, which lies in a constant and well-ascertained anatomical position, than a multitude of open mouths of its branches, whose position great good luck may possibly enable us to find, but which must always be problematical, and, in the majority of instances, will probably be inaccessible.

I have already said all I wish to say as to "retraction," and have no desire to add to the amount of "verbosity" which Dr. Herman so justly deprecates. After all, the central point in Dr. Herman's paper is to be found in the paragraph at the top of p. 447 (THE PRACTITIONER, April, 1907):—

"I hold the current teaching that it is uterine contraction which stops post-partum hæmorrhage to be correct. The only effective treatment of post-partum hæmorrhage is to bring about uterine contraction. Pressure is only a temporary means of suspending bleeding until the uterus has regained its contractile power."

In these sentences I begin to see the probability of our ultimate agreement; I maintain as strongly as Dr. Herman that the only final and permanent stop to post-partum hæmorrhage is uterine contraction, but such contraction, as he himself admits, is not "regained" at this, the crucial time, the time of acute danger from loss of blood, and I appeal to his own words, on the succeeding page, as the best confirmation of my contention as to the mode of action in such circumstances. "The cure for a tired uterus is the same as that for fatigue of any part of the body, sleep . . . after sleep . . . uterine action will return." It is true that he is there speaking of the second stage of labour, but surely the uterus may be as tired, if not more so, in the third stage as in the second. It is only natural that it should be so, having undergone more fatigue. The condition has not changed, it has only become more marked, and the remedy remains the same; sleep, and consequent recuperation for the uterus, stoppage meanwhile of loss of blood, by other means which do not involve its reawakening until the full benefit thereby to be obtained has accrued. We both believe in the "final" stoppage of hæmorrhage by the contraction of the uterus. Apparently we do not—yet—coincide as to the best means of bringing this about.

But it seems to me that things are improving. Already we have Dr. Herman's pronouncement that some of the previous teaching is "Stuff," "preposterously unpractical," and that a student, who should venture to put upon an examination paper what is the only logical outcome of the teaching of text-books. ought, without hesitation, to be referred. "Such a man," he says, "ought not to be at large; he is dangerous." Whatever I may have thought, I have never myself ventured to speak so strongly; this is decidedly encouraging. And one well-known authority on midwifery has confided to me that, in his forthcoming work on that subject, aortic compression with pelvic elevation will be taught as the one and only method to be advised; that midwives, under his tuition, are already being taught this method, and that the old series of "manipulations" are no longer to be taught in any institution under his control, except possibly for examination purposes.

Meanwhile, I receive letters from India, Australia, and all parts of the country thanking me for bringing this question up, expressing agreement with the arguments set forth, and, what is best of all, details of cases saved from uttermost danger by the procedure advocated.

One point only remains. Dr. Herman says, "The curious thing is that Mr. Bishop should write as if he thought compression of the aorta . . . was something new." It is still more curious that Dr. Herman should have formed any such idea; I have never suggested that it was new, simply because I have always known of the authorities to which Dr. Herman refers; in some of my papers, I have distinctly said so. Perhaps the pelvic elevation, in order to control the venous flow, which Dr. Herman thinks so unimportant, but which I am quite content to leave to the common sense of the profession, may be original, or it may not. It matters nothing. What is important is the truth, and trustworthiness; or the error, and untrustworthiness of the plan indicated. Let us not obscure this essential issue, by dragging in personal disputes as to priority or any other trivialities. The lives of parturient women, and the reputations of our professional brethren, are at stake; everything else is of no moment.

THE EVIDENCE AND CONCLUSIONS RELATING TO THE MULKOWAL TETANUS CASE.

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In the supplement to the Indian Gazette of December 1, 1906, there appeared a collection of papers referring to the Mulkowal tetanus accident, which occurred in the Punjaub in October 1902, in the course of the great campaign of anti-plague inoculations of that year. The papers include the report of the Indian Commission, those of the Lister Institute, to which the conclusions of the Indian Commission were referred for opinion, the comments of Mr. Haffkine on those reports, and extracts from the evidence of various officers, including that of Dr. Elliot, who inoculated at Mulkowal, and of the compounder who assisted him. The whole of the proceedings are not published, but the papers and reports issued are sufficient to enable the reader to analyse the evidence on which the conclusions are based. The facts narrated, and the conclusions arrived at in these papers, will form the basis of this review.

The results of the Punjaub inoculation campaign of 1902-03 were reviewed in THE PRACTITIONER of December 1905. It will be remembered that 505,849 persons were inoculated, and it was estimated that at least 12,917 lives were saved from plague during that season.

The Accidents which preceded the Mulkowal Case.— The commencement of the campaign, which had been organised very hurriedly, was attended by a series of accidents. The first of these included abscesses and four deaths from symptoms which remained undetermined, and one death from tetanus. They were demonstrated not to be attributable to the prophylactic fluid. They occurred in single individuals out of many inoculated from the same bottle. If contamination had been introduced into a bottle of prophylactic several weeks before in the laboratory, it would, in all likelihood in the fertile medium which the prophylactic has been proved to be, and at the temperature of the Indian climate, have multiplied and rendered each dose infective. The Commission investigated these accidents, and reported that the Punjaub officers acknowledged that the deaths were not attributable to the laboratory, and, with reference to the abscesses, the Commission affirmed, in addition, that, from the facts before them, it was impossible to connect the abscesses with the fluid rather than with defective inoculation. As regards the death from tetanus, they stated that they were "satisfied that the isolated case of death from tetanus cannot be ascribed to contaminated fluid." These conclusions exonerated the laboratory, and indicated that, in the *technique* of inoculation, there was something wrong, which was causing accidents, including tetanus. Later, it will be seen that the *technique* was defective, and did not follow the instructions of the laboratory. With the return to the correct *technique,* in the beginning of November after the Mulkowal case, there were no accidents attributable either to the laboratory or to the inoculations.

The Object of the Investigation into the Mulkowal Case.—The object of the investigation into the *Mulkowal case*, which was of a far graver nature than the preceding cases, was to find out where the contamination took place; was it in the laboratory, or on transit, or at the place of inoculation? And how did it occur? No other issue could be of importance in connection with the primary subject of investigation. We mention this, because the report, even in the abbreviated form in which it is published, deals with a mass of other material not in direct connection with that investigation.

The Facts regarding the Mulkowal Accident.—The circumstances of the accident were as follows:—107 people were inoculated at Mulkowal on 30th October 1902. Of these the first 19 were affected with tetanus. The symptoms appeared on the 4th and 5th of November, and the patients died between the 6th and 9th. The other 88 inoculated were unaffected. The first 19 were inoculated from the same bottle, and no one else was inoculated from it. The syringe, as originally prepared for the inoculations of that morning, was used for the first 19 persons. About half of the others were inoculated later on from the same syringe, but after it had undergone a certain superficial process of cleaning. The remaining half were inoculated with another syringe. No other appliances, used in the inoculation, such as lint, carbolic lotion, etc., were used exclusively on the 19 affected. Evidently the accident was connected either with the syringe or the bottle. An experiment was made to ascertain whether it was possible for a contaminated *syringe needle* to infect 19 persons, and, after the Mulkowal process of cleaning, to infect no more. This was done by the Lister Institute (Appendix G. of its report, experiment 2), which demonstrated that a syringe needle, contaminated by being momentarily dipped into a broth culture of tetanus, and then allowed to dry, sufficed to infect afterwards Haffkine's prophylactic fluid no fewer than 17 times, when this fluid was ejected through the syringe, the dose ejected each time being 1 c.c. On the syringe being filled with carbolic solution 1 : 20 for three minutes and emptied, as had been done by the Mulkowal operator, it became safe for use ; for, on filling it again with Haffkine's prophylactic, and testing the fluid in the same way as before the cleaning, the results were negative.

The possibility of infecting uniformly 19 persons by an impurity mixed up with the *fluid* is so obvious that it requires no demonstration.

In the event of the accident having been produced by a contaminated needle, which, as has been seen, is quite within the range of possibility, the source of that contamination is referable only to the inoculation *technique* followed in Mulkowal. If, on the other hand, the impurity which caused the accident came primarily from the fluid in the bottle, and not from the needle, the accident is referable either to Bombay, or to the period of transit, or to Mulkowal.

That it was possible, at Mulkowal, for a minute impurity falling into the bottle to cause 19 cases of tetanus is already evident from the experiments with the needle mentioned above. The same minute quantity of contamination getting into the fluid, instead of adhering to the needle, would have evidently sufficed to produce the same results. This was demonstrated by Haffkine's experiment with a small bit of straw dropped into a bottle of sterile broth (Appendix C., Indian Commission's Report), which was sufficient to permeate the bottle straightway, and to give at least 50 germs to each dose of fluid.

It was also demonstrated by Semple's experiment with a piece of straw, contaminated with tetanus and left to dry (bottle No. 4, experiments 6, 7 and 8). The straw was dropped into a bottle containing 30 c.c. of prophylactic and the bottle was shaken. Then the straw was removed. A culture, made with a droplet of the fluid, gave a typical growth of tetanus on the third day (experiment 8). Germs, therefore, were present in each droplet in the fluid in the bottle, when examined a few minutes after the impurity was introduced. Considering the number of droplets in 30 c.c. of the fluid, it indicates the large number of tetanus cases that could be produced under suitable conditions.

Semple's experiment (bottle No. 3, experiment 5), with a bottle of prophylactic contaminated with a droplet (a loopful) of a fluid culture of tetanus, is equally instructive. He inoculated immediately two guinea-pigs from the bottle, one with 1 c.c. and the other with 1½ c.c.; both contracted tetanus.

This shows that a small amount of impurity can uniformly permeate a fluid immediately so as to cause at least 30 accidents when the fluid is subjected to the swirling action of being drawn through the needle of a syringe. A "loopful" from a fluid culture contains infinitely fewer microbes than a small particle of solid dirt, that might fall into a bottle.

The feasibility of the source of the accident occurring primarily at Mulkowal is thus demonstrated.

The facts which established which of these possible alternatives, i.e., *contamination in Bombay, during the period of transit, or at Mulkowal, did actually take place.*—It was ascertained by experiment that the water agar prophylactic was a rich culture medium for the tetanus bacillus isolated at Mulkowal.

A fact of this kind necessarily has a decisive bearing on the condition of the fluid at the time of inoculation, the conditions being very different if the impurity had fallen into the fluid at Mulkowal, or before the fluid reached Mulkowal, as in transit, or in Bombay. If in Mulkowal, it would be used before a culture had grown. In the two other instances it would be used after the microbe had had time to grow. The differences would become manifest in the following way:—

1. By the absence or presence of a bad smell, which develops invariably when a growth of tetanus bacilli takes place.

2. In the symptoms of the patients, which would be either ordinary, such as those appertaining to tetanus cases caused by natural infection, or extraordinary, caused by the luxuriant multiplication of the germ and the production of the toxin in the fluid.

3. In the condition of the dregs of the bottle.

4. In the effect of the contaminated syringe when used afterwards on other patients.

As previously mentioned, if the bottle had been contaminated in the laboratory there would have been an abundant and very toxic growth.

Experiments by Semple and the Lister Institute were made under a great variety of conditions with the Mulkowal and other tetanus germs. In some cases the germs were taken by themselves, in others in association with other microbes. They were inseminated in water agar prophylactic of different manufacture, and were placed in bottles which were full of fluid, or only partly filled, or contained only a few drops. In all cases, a rich culture of tetanus and the characteristic smell were recorded, in Semple's observations after seven days, and in the Lister Institute's after three days.

What were the actual facts in the Mulkowal case?

(1) The bottle was examined before proceeding to inoculate, and the fluid had no smell. A perusal of the statement made by Dr. Elliot, the inoculating officer, and his assistant leaves no doubt about this most important fact. This evidence gives a decisive answer as to the condition of the bottle at the time, and it is as direct and unmistakable as can ever be obtained by any methods of investigation. This fact, moreover, does not stand by itself, but is confirmed by every other fact in the case.

(2) The reaction observed on the 19 persons was similar to that which occurs in average Indian clinical cases, when an infection is produced by an invisible impurity remaining in the tissues in an abrasion produced by a fall or other accident. The results would have been very different if the infection had been caused by 1½ c.c. of an extremely toxic and rich culture. Semple's experiment, Appendix B., experiments 1 and 2, and Appendix A., experiments 3 and 4, show that a tetanus culture when grown in the water agar prophylactic was several hundred times more toxic than that grown in an ordinary medium and acted much more rapidly. It is well known that the larger the dose of microbes and of toxine, the shorter the incubation period, and the more rapid the fatal result. (*Vide* Lister Institute Report, Appendix F. (*b*), experiment 2.)

(3) If contamination occurred in Bombay, or in transit, while the bottle was full of prophylactic, there would, as stated, have been a rich and toxic culture; if, at Mulkowal, where the bottle was emptied immediately after the contamination, the culture, if any, in the dregs would have been poor. The microbe might grow, if assisted by the accompanying anærobic microbes present in the dirt, but they would only grow in proportion to the extent that the other microbes absorbedt he air, and it is well known that, under these conditions, the culture loses much of its virulence and toxicity. Col. Semple, on examination of the bottle, 15 days after the accident, found in it not a rich and very toxic culture as he obtained when the tetanus germs grow in a closed full bottle, but a culture which was poor in bacilli, and several times less active than one grown in a full bottle. This fact bears out the previous one, viz., that the contamination had not been even a few days in the bottle, when full of the prophylactic; and excludes the whole period up to shortly before the inoculation in Mulkowal as the possible time of contamination.

(4) The next and last fact elucidated by the enquiry, which again coincides with and confirms the others, refers to the syringe.

The special experiments, made by the Indian Commission and the Lister Institute, demonstrated that had bottle 53 N. contained a tetanus culture which had grown in a full bottle of prophylactic, the syringe, after the Mulkowal cleaning, could not have been rendered safe for the persons inoculated from other bottles. Experiment 2, Appendix B., of the Commission's Report, shows that 17 animals out of 19, operated on with such a rinsed syringe, had tetanus. An analogous experiment, but only on cultures, was made by the Lister Institute (Appendix G. of the Institute's Report, experiment 1) and the results were the same.

On the other hand, if a syringe comes in contact with a poor culture, or if only the needle of the syringe is contaminated, the experiments (Appendix A., experiment 2 of the Indian Commission; Appendix G., experiment 2 of the Lister Institute) demonstrate that the syringe can be easily rendered safe by slight cleaning, such as was practised at Mulkowal. The fact that, at the latter place, none suffered who were inoculated with the syringe subsequent to the first bottle, indicates that the bottle did not contain a culture even a few days old.

The bottle was sent from the laboratory to the Punjaub 26 days before the accident. As it takes only a few days for a culture of tetanus to develop in the prophylactic, the above facts point to the exclusion of the Bombay and transit period *up to at most three days before opening the bottle for inoculation.*

There was on the cork and sides of the bottle a label which gives instructions as to the opening of the bottle, and which must be torn before the cork can be removed. At Mulkowal this label was found intact, and the cork tightly secured in the bottle. This excludes even the three days' interval preceding the opening of the bottle at Mulkowal, as the time when contamination could have got into the bottle.

The above facts decide the important question as to the stage at which contamination took place, and clearly demonstrate that it took place at Mulkowal.

It remains to examine in detail the procedure at Mulkowal, and particularly that part of it which deals with the handling of the syringe and the bottles.

Instructions regarding the inoculations were given in a *Punjaub Plague Manual*, 1902, which was issued to the plague operators by the local authorities shortly before commencing the campaign.

According to this Manual, the preliminary treatment of the syringe and needle is entrusted to the inoculator himself, who is instructed to sterilise the needles in hot oil heated to 160° C. The syringe itself is to be filled with carbolic lotion 1 to 20 for a period of 24 hours previous to use. These procedures conform to the essentials of effective sterilisation.

The instructions as to the treatment of the bottle and fluid are, however, as follows :—"The opening of the bottles of prophylactic, and the filling of the syringes are the chief duties of the compounder," that is, an assistant very little different from an Indian villager. He is to carry out these duties in the following way :—"The compounder, in proceeding to open a bottle, must shake it well, then dip its neck into carbolic lotion 1 in 20, and then with a pair of dissecting forceps, which when not in use are to be kept in carbolic lotion, remove the cork." "If there is a great press of work the duty of actually opening the bottle may be entrusted to any intelligent person, care being taken to see that he observes the above instructions carefully." It is explained in the instructions that the intelligent persons to help in the work are to be selected from the villagers gathered together for the inoculations.

These local rules were substituted for the instructions issued by the laboratory which were as follows :—

The sterilisation of the needle is to be done either with the syringe by means of hot oil or 24 hours soaking in carbolic acid, or else "separately from the syringe, by boiling in a cup, or in a test tube for 10 minutes before use. The procedure is to be repeated every time the needle happens to fall on the ground during the operations, as the ground dust contains dangerous micro-organisms. After boiling, the needle is not to be touched with the fingers, but taken out with a pair of forceps, the points of which have just been heated in a lamp."

Next the method of opening the bottle is thus described in the laboratory instructions.

"Dissecting forceps are to be used for removing the stopper, the branches of the forceps are heated in the flame of a spirit lamp, immediately before use, and guarded from contact with any unsterilised object afterwards. The stopper and neck of the bottle are then passed several times through the flame of a lamp, so as to cause very slight singeing of the stopper ; and the latter is withdrawn with the heated forceps. After the bottle is opened, any contact between its mouth and other, unsterilised, objects is to be carefully avoided ; and if contact inadvertently occurs, the mouth is to be heated again in the flame, to disinfect it." During the Mulkowal operations, the instructions of the local "Manual" were followed, and bottles and forceps were asepticised by being dipped in carbolic lotion. It should be noted that tetanus spores require 15 hours soaking in 5 per cent. carbolic acid to be rendered harmless ; and the procedure prescribed is accordingly useless for the purposes of sterilisation.

Coming now to the actual circumstances in which the Mulkowal inoculations were performed, the Commission found that an important incident occurred during the opening of the bottle in question. The compounder, in removing the india-rubber cork, dropped the forceps on the ground when the cork began to move, and after picking it up he "swished the forceps in the lotion and then pulled out the cork with it."

It was found also that it was not an infrequent occurrence, on these occasions, for the forceps and the corks to fall on the ground, and be treated in the same way.

The above facts show that, although the accident was feasible by an infected needle, as well as by a contaminated fluid, the details of the operation, as performed at Mulkowal, point to the faulty handling of the bottle and the contamination of the fluid thereby, as having been the primary cause of the calamity.

How the facts were dealt with by the Indian Commission and the Lister Institute.—The Commission began by excluding the possibility of the needle or any other instrument or appliance producing the accident; the reason being that the accident was confined to the use of bottle 53 N. The Lister Institute, by experiment 2, Appendix G., demonstrated that a contaminated needle infected at least 17 consecutive injections, and, after rinsing, appeared free from further infection. This at once renders admissible that a contaminated needle may infect every person out of a bottle of some 20 doses, and, after rinsing, not to infect any one inoculated subsequently. Turning to the fundamental question as to how, when, and where contamination actually took place, we find that, in treating this subject, the Commission do not bring to bear on it all the facts which the inquiry has brought out, and those facts which they do bring out are dealt with in a manner which is fallacious. The following is the Commission's judgment regarding the absence of smell in the bottle :—"No doubt the presence of tetanus, at any rate in a pure culture, may be detected by one experienced and on the alert, by its characteristic smell; our personal observation, however, leads to the conclusion that all bottles of plague prophylactic have a peculiar and not agreeable smell, so that Dr. Elliot, in the circumstances, in which he was working, may well have failed to specialise a tetanus smell. The failure to perceive a smell is undoubtedly a fact which calls for consideration."

It does not require a specialist to detect whether a bottle has a smell or not. Here Dr. Elliot is represented as having before him the problem of recognising the nature of a tetanus smell, whereas he had only to ascertain whether the bottle had a smell or not, and he ascertained beyond all doubt that there was no kind of smell. Moreover, the statement that all bottles of prophylactic have a peculiar and not agreeable smell is incorrect. The *broth variety* of the prophylactic possesses a slight odour of broth and carbolic acid, but it was not in use in the Punjaub at the period of the Mulkowal accident. The *agar variety* has not the faintest smell, and it was this alone that Dr. Elliot was dealing with at the time. Dr. Elliot, in order to encourage the villagers to be inoculated, was going to be inoculated himself first, and it is natural that, in these circumstances, he would be particular and much on the alert as to the fluid he was using. Indeed, the Government of the Punjaub reported immediately after the accident to the Government of India as follows :—

"Dr. Elliot, the inoculator, adopted every necessary precaution to guard against accident, and he had no reason to suspect the fluid on account of its smell or appearance, or any other account whatsoever. He was so sure of its harmlessness, that he, in a practical way, signified to the people his own readiness to be inoculated with it.

"Before commencing to inoculate at Mulkowal on the 30th of October, he offered to be inoculated himself in order to gain the confidence of the people, and had actually bared his arm for the operation when the people said they would be inoculated without this."

The compounder, who assisted Dr. Elliot, stated :—"Dr. Elliot took out the bottle from the box and gave it to me ; that was the first thing in the morning. I was to open the bottle . . . . I took out the cork with the forceps. I kept the cork with the forceps in my hand, and Dr. Elliot smelt the bottle."

Dr. Elliot's statement was that "I began my operations that morning with 53 N. I smelt the bottle and noticed nothing ; nothing interfered with my power of smelling. I was on the look-out, as I had discarded a bottle from the same box the previous week . . . . I have a good sense of smell, and I am positive the bottle did not smell."

Dr. Elliot mentioned also that the water agar prophylactic, which he used in the Punjaub, unlike the broth prophylactic which he used in Sindh and in the Bombay Presidency, had no smell. This, he said, made it easier for him to detect any bottle which might possess the slightest smell.

His explanation was confirmed by the following test :—

The Commission, in their cross-examination, placed before Dr. Elliot three bottles. They did not inform him what these bottles were, but asked him to examine them as to their condition. Dr. Elliot smelt the bottles, and had no difficulty in differentiating them. He pointed out one that had no smell, a second that had a faint odour, and the third he rejected as being unfit for use on account of its bad smell. When asked to specialise this smell he said he was unable to do so, and, when asked if he knew the smell of a tetanus culture, he replied that he did not. Of the three bottles, the first which had no smell proved to contain water agar prophylactic, the second which had a faint smell contained the broth variety, and the third was the original Mulkowal bottle.

This evidence is very clear. It shows, first, that at the time of the accident at Mulkowal the bottle did not contain a culture of tetanus even a few days old, and secondly, that both culture and smell, which subsequently developed, were quite sufficient if they had been present at the time of opening the bottle to have caused the inoculator to reject it. It will be remembered that the inquiry supplied facts confirmatory of both these results, viz., of the absence of culture at the time of the operation, and of the development of the germs subsequent to the emptying of the bottle. The absence of culture and toxin, at the time of the operation, was corroborated by the slow course of the disease among the 19 persons infected, and by the facility with which the syringe was freed from contamination. The development of the germs subsequent to the emptying of the bottle was shown by the kind of culture, which was found in it on examination, and which indicated a growth developing under ærobic conditions, and not the rich and very toxic culture which was found to develop in a few days in a full bottle of water agar prophylactic. After having considered the circumstances relative to the odourless condition of the bottle, the Commission say " it only remains to consider the value of the inference based on the tardy manifestation of the disease." On this they bring to bear the following data :—

The fact that the tetanus in the Mulkowal cases took about the same time to develop as is observed in accidental cases caused by natural infection.

That, in natural infection, the disease is produced by the introduction of microbes without an additional dose of ready made toxine, which has to be elaborated by the microbe before the symptoms can be caused, and,

That experiments on animals show that an additional dose of ready made toxine hastens the appearance of the symptoms. The Commission in their own experiments had evidence of this (expt. 1, Appendix B.).

In the Lister Institute's report (Appendix F. (*b*), expt. 6) we find an experiment, in which the bacilli and spores from 1 c.c. of a month old tetanus culture were separated by filtration from its toxic supernatant fluid. The whole mass of the microbes was then injected into a guinea-pig, but caused only a slight tetanus from which the animal recovered. The effect of the toxine was then tried on guinea-pigs with the result that—

(*a*) $\frac{1}{500}$ c.c. of the toxine killed the animal in seven days.
(*b*) $\frac{1}{400}$ c.c. killed another animal in six days.
(*c*) $\frac{1}{300}$ c.c. killed another in five days, and
(*d*) $\frac{1}{200}$ c.c. and $\frac{1}{100}$ killed in four days. (F. (*b*), expt. 2).

The result would certainly have been still more marked if the animals, instead of being injected with toxine alone, had been injected with toxine and doses of microbes, as would have been the conditions in the Mulkowal cases had bottle 53 N. contained a tetanus culture several weeks old.

There is recorded the case of Dr. Nicolas, Professor Arloing's assistant, who, in the course of experiments, accidentally pricked his finger with a needle moistened with tetanus toxine. He developed tetanus in 4 days. Considering that the Mulkowal cases would have received $1\frac{1}{2}$ c.c. of an extremely active toxine their incubation period would have been much hastened compared to Dr. Nicolas's case.

Such was not the character of the disease in the Mulkowal cases. On the contrary their incubation period lasted longer. The obvious conclusion is that the bottle contained no free toxine, and the dose of tetanus germs, which were conveyed with its fluid, was insignificant and similar to that which causes natural clinical cases.

The conclusion drawn by the Commission is of quite an unexpected nature, and is to the following effect :—"There can be no appeal to clinical experience" ; and "we cannot estimate the exact import of the duration of the incubation in the Mulkowal cases."

It may be added that the Commission dwelt only on the incubation period, and omitted the inference to be drawn from the duration of the disease itself, which points as strongly to the comparative small amount of infection conveyed to the Mulkowal patients. Similarly they dwelt on the effect of the toxine alone, and omitted the effect of the very large mass of germs, which would have been present in a bottle contaminated in Bombay, and which would have had a similar effect in shortening the incubation period and the course of the disease, as the accumulated toxines would have had.

The facts, as they stand, point to a very definite conclusion, viz., that there was no toxine and no culture of germs in bottle 53 N. at the time of inoculation, and the Commission are certainly wrong in stating that no conclusion can be drawn from those facts.

The Commission then say, "This concludes the considerations favouring the view that the contamination was introduced at Mulkowal." As has been mentioned already, this is not the case, as there are other considerations pointing to the contamination having occurred at Mulkowal. They have omitted to take into account the evidence derived from the condition of the syringe, after it was used for bottle 53 N., and from the condition of the fluid in this bottle when it was examined a fortnight after the accident.

As stated above, in an experiment made by the Commission with a bottle of prophylactic, which had been infected seven days previously, it was found that a syringe, filled with it and then emptied, could not be rendered safe by the cleansing process employed at Mulkowal. For, on being refilled with sterile broth, and used for injecting animals with the same doses as were employed at Mulkowal, the syringe gave tetanus to 17 out of 19 animals.

This experiment represented the conditions which would have existed at Mulkowal, if bottle 53 N. had been contaminated in the Bombay laboratory; and it clearly shows that if such had been the case, the persons, who were afterwards inoculated with the infected syringe from another bottle, would have taken tetanus.

On the other hand, in an experiment which was made by the Commission, and in which a tetanus culture several hundred times weaker was taken, and in another, made by the Lister Institute, in which no culture was used but the syringe needle alone was infected and had conveyed tetanus germs in 17 preliminary injections, the cleansing done at Mulkowal proved to be sufficient to free the syringe from tetanus. The absence of tetanus in the people inoculated at Mulkowal, after the first bottle, and subsequent to the cleansing of the syringe, points to the fact that that syringe had not come in contact with a rich culture of a bottle contaminated in Bombay, but with a much smaller amount of impurity, as would have occurred in an accidental contamination of the bottle, and before tetanus germs had sufficient time to form a rich culture in it.

Referring to these facts, the Commission state that "the experiments that have been made by Lieut.-Colonel Semple, appended to this report, show, we think, that the argument is so far well founded, that where a fluid containing a culture of tetanus is used, the syringe, even after it had been washed out with a 1 in 20 solution of carbolic acid, may retain enough contamination to affect the fluid with which it is subsequently filled. The extent, however, of its influence varies according to the richness of the growth between fatal results and absence of all manifestations. There are no means of forming an opinion as to the precise richness of the growth in the bottle 53 N. beyond Lieut.-Colonel Semple's view that it was a poor growth in comparison with that which he used in his experiments No. 1 and 2 in Appendix B.[1] We therefore are unable to accept the argument that the freedom from tetanus of those persons, subsequently inoculated, shows that the specific contamination could not have entered the syringe, and, after full consideration, we think the contamination was in the fluid."

It is obvious that the Commission do not draw the conclusion, to which the facts under consideration point. The facts do not point to any conclusion as to whether the contamination was or was not in the fluid ; for in both cases, if the amount of contamination was *small*, whether it was on the needle alone, or in the fluid, it was proved that the washing of the syringe protected effectively the people subsequently inoculated. The facts under consideration point to the conclusion that there was no *rich* culture of tetanus in the fluid at the time of the Mulkowal accident, as would have been the case if the fluid had been contaminated even a few days before the accident, consequently the bottle was not so contaminated.

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[1] That is, the experiments in which a 7 days' culture of tetanus in water agar prophylactic contaminated the syringe to such an extent that, after the cleansing, it infected 17 out of 19 animals.

The Commission also treat the bottle 53 N., when seen by Lieut.-Col. Semple 15 days afterwards, as being in the same condition as it was at the time of the accident. They were misled by an experiment made by Lieut.-Col. Semple, which was afterwards proved to be incomplete. The Lister Institute ascertained that germs of tetanus, left in the few drops of fluid in the bottle, would not remain in an unaltered conditon, but would multiply to a certain extent if, as is always the case in Nature, the impurity contains also other microbes. This, of course, only confirmed that which was already known to every bacteriologist concerning the behaviour of these germs.

Lastly, the Indian Commission has omitted to consider that the poverty of the growth found in bottle 53 N. has an important bearing on the question as to the time of the contamination of the fluid. As mentioned before, the Mulkowal and other tetanus germs, growing under the conditions obtaining in a *full* bottle of water agar prophylactic, produced in a few days a culture of exceptional richness and toxicity. The same germs, on the other hand, when left in a few drops of fluid remaining in a bottle, either do not give a culture at all, as was thought to be the case by the Commission, or give a poor culture of weakened toxicity. It was just such a poor culture, which was found in bottle 53 N. 15 days after the accident, and it showed that the contamination took place at the time when the bottle was emptied of its contents ; and that the germs had no time to grow while the bottle was still full of fluid.

The final conclusion arrived at by the Commission was that bottle 53 N. was contaminated in the laboratory at Bombay, and they give three stages in the preparation of the bottle, in one of which they say the contamination may have taken place. This conclusion is not supported by the facts brought out by the enquiry. That misgivings were felt by the authorities is evident from the fact that the matter was submitted for re-examination and report to the Lister Institute.

How the matter was dealt with by the Lister Institute. —The Lister Institute report on the subject of the investigation and on their own experiments as follows :—

"From consideration of the evidence, and, in the light of experiments (*vide* Appendix F.), the Institute agree with the Commission that, in all probability, the tetanus was at the time of the inoculation in the fluid contained in the bottle, but the fact that a bottle presumably tightly corked (*vide* Dr. Elliot's evidence) should contain enough tetanus growth to destroy 19 people, and yet not be accompanied by sufficient smell to arouse the suspicion of Dr. Elliot, who, according to his evidence, remembers smelling this particular bottle, is difficult to comprehend."

To analyse this statement, it should be borne in mind that it was subsequently explained by the Institute that *growth* of the tetanus impurity, which is always accompanied by a bad smell, was, in their opinion, not necessary for the causation of the Mulkowal accident, and that the possibility of the contamination having effected an entrance at Mulkowal, without having time to become a growth, could not be excluded. The antecedent assumption, therefore, that the contamination was in bottle 53 N. in the form of a *growth* or culture begs the question. The obviously correct deduction from the fundamental fact under consideration is that the absence of smell is a proof that the tetanus germs had had no time to develop into a growth or culture, and further, that any other deduction was inadmissible.

The Institute acknowledged later that the argument contained in this important statement was in favour of the contaminating not having occurred at Bombay, but the form given by the Institute to their pronouncement obscures that conclusion.

Dealing with the condition of the syringe in the Mulkowal operations, the Institute dwell on the number of germs required to start a tetanus culture, as compared with the number required to cause tetanus. These numbers may or may not be different; the question possesses, in the present instance, a purely academic character, for the Institute had before them experiments, made by themselves and by the Indian Commission, which demonstrated that the syringe, when contaminated with a prophylactic in which tetanus has grown, retained after the cleaning a sufficient number of germs to infect *both cultures and animals;* but they abstain from pointing out the conclusion which follows from the fact that, at Mulkowal, the syringe was so slightly contaminated, that it caused no further infection after its cleaning.

Turning to the fact of the lengthy incubation period in the Mulkowal cases, it was mentioned that the Indian Commission refused to draw from it the obvious conclusion that bottle 53 N. did not contain a culture and toxine of tetanus.

The Lister Institute, although their own experiments (Appendix F. (*b*), experiments 6 and 2) were very conclusive on this question, nevertheless, pass over the matter in silence.

Lastly, the fact in regard to the poverty of the growth found in bottle 53 N., as contrasted with the richness and toxicity of the growth, which the Institute and the Indian Commission observed to take place in a full bottle, was also passed over in silence.

As the final result of their investigation, the Institute in their report conclude that in all probability the contamination was in the fluid. This is not a luminous conclusion. The main question was not whether the contamination was in the fluid or in the needle, but where and when the contamination occurred. That is the vital question, and the case is not completed by stating that the contamination was in the fluid. Everyone agrees that, in all probability, the contamination was in the fluid rather than the needle, but the whole importance of the enquiry rests on the time and place of the contamination of the fluid. On this point the inquiry furnished facts for a very definite answer, and that answer is that the bottle was contaminated with tetanus germs at Mulkowal at the time of opening, and that it was brought about by the omission of the technique necessary for sterilising the forceps, cork, and mouth of the bottle.

ON RETINAL EMBOLISM, WITH TWO CASES IN WHICH A NORMAL STANDARD OF CENTRAL VISUAL ACUITY WAS RETAINED.

By C. O. HAWTHORNE, M.D., M.R.C.P.,

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[With Plate XVIII.]

RETINAL embolism is an event which, in the great majority of cases, is signalled by the occurrence of sudden and complete blindness, and this either remains as a permanent condition, or undergoes, in course of time, some slight measure of favourable change.

When this sudden loss of sight has been preceded, on one or more occasions, by temporary visual disturbances or interruptions, the diagnosis of embolism comes more or less definitely under suspicion; and this applies, even although blocking of the central artery is made certain by ophthalmoscopic or other evidences. In such circumstances, thrombosis, endarteritis, and even arterial spasm, have been offered, either alone or in combination, as factors responsible for the vascular obstruction. The suggestion has even been made, that it is in these causes, the majority, if not indeed the whole, of the cases labelled in clinical descriptions "retinal embolism," must find their true explanation. There is, however, reason to suspect that this statement is set in terms too sweeping, as, apart altogether from clinical observations, there is fairly distinct pathological evidence that true embolism of the retinal artery does undoubtedly occur. Whether, in any individual case, this, or alternatively, some other cause of arterial obstruction, is in operation, must be decided by a study of the whole of the attendant circumstances. The question, as a pathological problem, is one of great interest, and its relationships in this respect have recently been very fully discussed by Mr. George Coats,[1] Curator to the Royal London Ophthalmic Hospital, in a paper based upon a series of exact histological observations. As a clinical problem, however, the discussion can hardly be of more than academic value, for, given cessation of the blood supply, however produced, the functions of the retina are likely to be permanently damaged, and this, even although the circulation should be restored, after a comparatively brief interval.

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[1] *Royal London Ophthalmic Hospital Reports*, October, 1905.

In the first of the two cases here recorded (p. 819) the diagnosis of embolism can scarcely be questioned. The dramatic suddenness of the visual loss, the existence of mitral disease, and the ophthalmoscopic facts, taken together, hardly admit of any other interpretation. In the second case there may be reasons for a less confident conclusion. One of these is the absence, so far at least as the patient's judgment goes, of an abrupt or instantaneous character in the onset of the visual defect. Another is the non-existence of any evidence of cardiac or vascular disease from which an embolus could take origin. Yet the case is, beyond doubt, one of arterial occlusion. Hence embolism, thrombosis, and endarteritis appear to be the only possibilities, for spasm *per se*, always a somewhat speculative suggestion, can hardly be proposed as an explanation of arterial obstruction sustained to such a point as permanently to destroy the function of the greater portion of the retina. Now of these three available causes, the comparative youth of the patient (28 years), the absence of any evidence of arterial disease, and the freedom of the personal history from any hint of syphilitic infection, render disease of the wall of the retinal artery in the highest degree improbable. There remain, therefore, thrombosis and embolism. Each of these is not without its difficulties. Against embolism is the absence of any manifest source from which an embolus could readily arise. Against thrombosis are all the circumstances which render endarteritis improbable, for arterial thrombosis is usually, if not invariably, secondary to disease of the vessel wall. Even if the latter consideration be disregarded, it would have to be admitted that a thrombus, formed *in situ*, was a demonstration of an abnormal tendency to blood coagulation. And if this could display itself in the retinal artery, it might equally well do so in some other part of the circulatory apparatus, say in one or other of the chambers of the left heart. The supposition of primary thrombosis may, therefore,

PLATE XVIII.

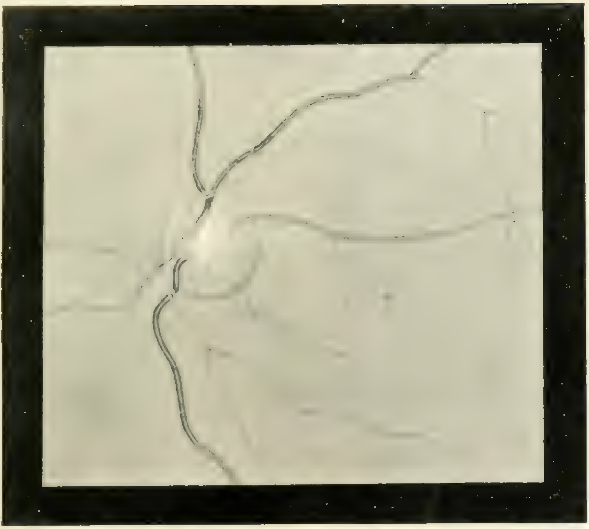

Fig. 1.

Fig. 1.—*Fundus Oculi shewing large Cilio-Retinal Artery supplying Macular Region, and Branches of Central Artery much reduced in calibre. Some of the latter have "white lines" along their course, and one, with a downward and inward direction, is practically empty.*

be made to apply as readily to an embolus as to a thrombus. as the cause of obstruction of the retinal artery in a patient free from all evidences of cardiac and vascular disease. Granted this, it may be asked whether any cause for such primary thrombosis can be suggested. Now the examination of the patient reveals no sign of disease other than an anæmia of the chlorotic type. But, although perhaps not very frequent, thrombosis is a recognised complication of chlorosis. Occurring mostly in the veins, or in the intracranial venous sinuses, it does sometimes happen in the arteries. There can thus be little hesitation in admitting that it may also be possible in the chambers of the heart. But a thrombus formed in the heart may be detached, and, if swept from the left auricle or ventricle, may be carried, for example, into the central artery of the retina. Hence there is no reason why a block in this artery, occurring in association with anæmia, should not be due to embolism. In the absence of endarteritis, the block must mean either embolism or thrombosis. And, on the whole, it is perhaps easier to believe that anæmia, with a resulting measure of cardiac dilatation, should give rise to an intra-cardiac thrombosis, than that it should produce an arterial thrombosis apart from primary disease of the vessel wall. In a previous paper,[1] two cases, exactly similar in this respect to Case II. (p. 820), have been reported, and whichever of the two views stated above may be accepted, it is manifest that, among the thrombotic possibilities of anæmia, must be reckoned the occurrence of obstruction of the central artery of the retina, with the clinical consequences associated with the diagnosis of retinal embolism. In the individual case now under discussion (Case II.), the description of the onset of the visual loss, though far from conclusive, may be said to favour thrombosis *in situ* rather than embolism. This comment, however, does not touch, or at least is less appropriate to, the previous records, and, in any event, it must be judged together with the general rebutting evidence already related. Hence the balance of evidence, though allowing some room for uncertainty, seems to favour the diagnosis of embolism, and it is certain that, for ordinary clinical classification, the present case exactly accommodates itself to that diagnosis.

Assuming then that each of the two cases here recorded is an example of embolism of the central artery of the retina, the most remarkable feature is the retention, in each instance, of a full measure of central visual acuity. Indeed, this peculiarity remains without diminution of interest, even if a thrombus or other agent is substituted for embolus as an explanation of the arterial obstruction. The experience is only too general, that such obstruction carries with it complete loss of sight in the affected eye.

Occasional cases have been recorded, in which, not the whole field, but some limited part of the periphery only has been lost, this being due, presumably, to obstruction, not of the main artery, but of one or more of its terminal branches. On the other hand, there have been instances, in which, with retention of the greater part of the field, there has been a permanent central scotoma, as the result, apparently, of the entry of the obstructing clot into a macular artery, the other vessels of the retinal circulation remaining pervious; and this, as it involves loss of central vision, is almost as disastrous as obstruction of the main vessel. In the present cases, this latter position is reversed. Each patient

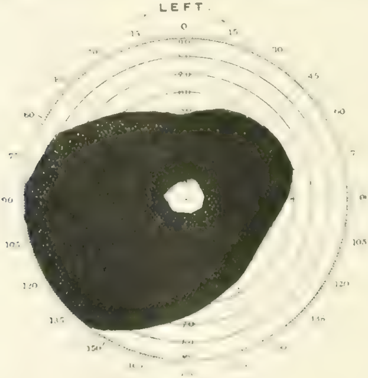

Fig. 2.

Fig. 2.—*Visual field from Case the fundus of which is represented in Fig* 1. *The field is blind except for a small area surrounding the fixation point; here vision is* 6/6 *and J*1.

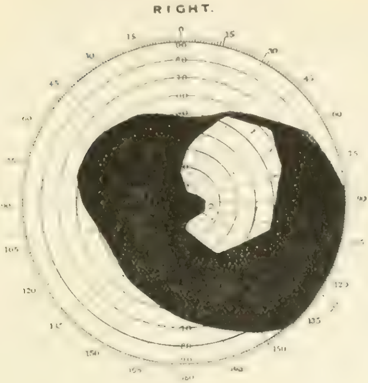

Fig. 3.

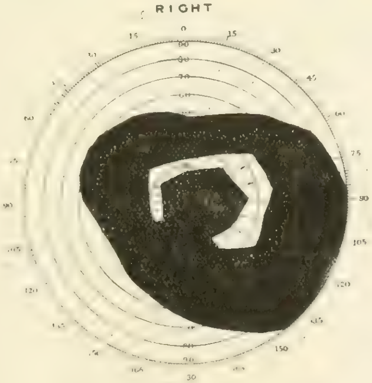

Fig. 4.

Figs. 3 and 4.—*Visual fields from Cases of Retinal Embolism. The greater part of each field is obliterated, but vision is still possible in a limited area, which, however, is eccentric, and does not include the possibility of central vision. In taking the visual field of an eye deprived of central vision, it is not easy to secure fixation, and hence the definition of the field may be more or less inexact. But if the patient is instructed to place his index finger on the fixation point of the perimeter, and is told to look steadily at the finger, a confident field can, with a little patience, almost invariably be secured* has perfect central or direct vision, but the field is restricted to a very limited area surrounding and including the fixation point. Manifestly, therefore, though the blood supply has been cut off from the greater part of the retina, it has remained effective to the macular region. It is perhaps not very uncommon to find, in cases of retinal embolism, that a small part of the field is retained, this being due to a supply of blood reaching, in one way or another, the corresponding part of the retina. The source of this supply may be either a branch from one of the ciliary arteries (cilio-retinal artery), or a branch which takes origin from the central artery in its course in the trunk of the optic nerve.

As the obstruction, whether embolus or thrombus, usually blocks the main artery at the level of the optic papilla, the circulation in a branch, such as the one just described, is not affected, and, consequently, when such a vessel exists, the blood supply, in spite of the block in the central artery, may be continued to a limited portion of the retina. Another possible explanation of the persistence of some part of the field of vision in cases of retinal embolism may be, as suggested by Coats, the establishment of capillary anastomoses between the ciliary and central visual systems. In Figs. 3 and 4 are shown two perimeter tracings from cases of retinal embolism, and each exhibits a small eccentric visual field, the existence of which is to be explained in one or other of the fashions just described. Neither in these nor in other similar instances, as a rule, does the retained portion of the retina include the macular region, and there is, therefore, loss of central vision. In each of the two cases here related, however, there was retention of central vision, and the explanation was evident on ophthalmoscopic examination. As is seen in Fig. 1 (and the two cases presented a practically identical picture), the blood supply of the retina is here not wholly dependent on the central artery. Towards the outer part of the disc is another artery, which is at once recognised as a cilio-retinal vessel, a branch, that is, not of the central artery of the retina, but of the ciliary system. And, manifestly, while the branches of the central artery are greatly reduced in calibre, the blood stream within them having been brought to a standstill and the distending force of the blood pressure being therefore absent, the cilio-retinal vessel is of relatively large calibre, and is in the enjoyment of an active circulation. It is to the presence of this vessel, and to its distribution to the macular region, that retention of central vision is due, while, in consequence of obstruction of the central artery, the whole of the peripheral field is blotted out. The presence of a cilio-retinal artery is, of course, not very uncommon, but it is not always so well developed as here, nor so intimately related to the macular region. Unfortunate, therefore, as these two patients are as victims of retinal embolism, they are to be congratulated upon their escape from the most terrible consequences of that condition, namely, complete and permanent loss of sight in the affected eye. They retain, on the contrary, perfect function in the most important part of the visual field, and for that happy circumstance, they are indebted to the arrangement which has provided each of the affected eyes with a special blood supply to the macular region.

In a previous communication,[1] cases of retinal embolism, homonymous hemianopsia, and optic neuritis, were reported in four women, the subjects of anæmia, and it was argued that these visual disturbances had, in all probability, a common basis in the shape of the thrombus formation which, without question, may complicate the anæmic state, or at least that form of anæmia known as chlorosis. The two cases, which form the basis of the present paper, are, perhaps, mainly of interest as illustrating how, as the result of an unusual vascular supply, an obstruction of the central artery may fail to prejudice central vision. But the second case has this further value, namely, that it emphasises the possible range of the consequences of anæmia through the occurrence of intra-vascular blood-coagulation.

CASE RECORDS IN ABSTRACT.[2]

Case I.—Sudden Loss of Sight in Left Eye, Return of Full Visual Acuity but Permanent Contraction of Visual Field; Cilio-Retinal Artery in Affected Eye. Patient the Subject of Mitral Disease.

Wm. P., æt. 24, was in his usual health when, in November 1903, while at business, he suddenly felt dizzy and exclaimed:

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[1] *Loc. cit.*

[2] For the opportunity of observing these cases, I am indebted to the courtesy of my colleagues, Mr. Britten Archer and Mr. Ernest Clarke.

"I have gone blind in my left eye." Seen two days after this occurrence, vision was less than 6/60, and there was some haziness of the edges of the optic disc and an appearance of œdema over the whole of the central region of the fundus. The visual acuity gradually improved and ultimately reached 6/6, and the fundus assumed the appearances represented in Fig. 1, the state of the visual field being shown in the corresponding chart (Fig. 2). Patient was myopic to 2·0 dioptres, but with the corresponding spherical lens was soon able to read 6/6, and J1 at twelve inches. Examination of the chest detected a systolic murmur over the cardiac apex, with accentuation of the second pulmonic sound ; patient had an attack of rheumatic fever in 1901. When seen again, in January 1906, the facts showed no alteration in any respect.

Case II.—Extreme Contraction of Left Visual Field with Retention of Acute Central Vision ; Optic Disc pale, with slight evidence of Neuro-Retinitis and Cilio-Retinal Artery. Patient anœmic, but otherwise free from disease.

Rachel C., æt. 28, suffered from whooping-cough at the end of 1903, and, in December of that year, noticed a sense of "stars shooting and brightness" before the left eye, and followed after a few days by "dulness of sight" in this eye ; this "dulness" has proved persistent. When seen in February 1905, V.A. was 6/6 in each eye, the left field was reduced to a small area around the fixation point, and the ophthalmoscopic appearances were practically identical with those figured from the previous case (Fig. 1). Beyond manifest anæmia, no evidence of disease could be discovered. The personal history was one of good health with some want of vigour during the last year or two. Patient the mother of three healthy children ; one miscarriage.

General condition much improved under the administration of iron, but no alteration of visual field or ophthalmoscopic appearances to June 1906.

THE TREATMENT OF DROPSY BY DEPRIVATION OF SALT.

By C. F. HADFIELD, M.A., M.D. (Cantab.), M.R.C.S.,
Malvern.

In the study of the pathology of œdema, continental observers have obtained very striking results, by controlling the amount of sodium chloride in the diet of dropsical patients.

The discovery that œdema could be produced in a suitable patient by the administration of certain doses of salt, early led to the more important observation that deprivation of salt could reduce a pre-existent œdema. In carrying out this treatment, the efforts of the French physicians have met with great success; and theories of far-reaching significance have been founded on their observations.

Widal has found that many cases of parenchymatous nephritis do not secrete as much chloride in the urine as they take by the mouth. This phenomenon he describes as "impermeability of the kidney to sodium chloride." The impermeability is never absolute, but is often nearly so. It greatly varies not only in different cases, but at different stages in the same case. This varying retention of chlorides in the organism he has been able to associate with the presence or absence of œdema.

In 1903, Widal and Lemierre found that, by adding salt to the diet of a patient suffering from parenchymatous nephritis, they could induce œdema, or increase it if already present. As the salt was diminished, the œdema commenced to subside.

In the same year, Widal and Javal tried the effect of reducing the amount of salt in the diet of such patients. This they have done again and again; and the results they have published show surprising success. In one of their cases, the patient, who was suffering from parenchymatous nephritis, was before treatment quite unable to take any solid diet without increasing not only the albumen in his urine but also the amount of œdema. An examination of his urine showed a marked retention of chlorides. He was treated during a period of 72 days; during this time, his diet was changed, in various ways, no less than nine times. On an ordinary diet containing meat, the œdema rapidly increased. If now all possible salt was excluded, the diet remaining otherwise the same, the œdema diminished, and the patient became rapidly better. A milk diet also suited the patient well, but if salt was added, as little as ten grammes a day sufficed, œdema and increased albuminuria at once recurred. This could now be cured by changing to a saltless meat diet, or by discontinuing the added salt in the milk. By weighing the patient, it was found that, on salt diet, he retained water in the body to the amount of six kilogrammes. It was noticed that, when salt was given, the body weight began to increase several days before the œdema became visible. Widal attributes this to a general hydration of the tissues, which, while not sufficient to be perceptible to the eyes and fingers, may nevertheless involve the retention of a very considerable amount of water. He describes this as the stage of pre-œdema; and its existence is, in his opinion, of very great significance. He thinks that many of the bad symptoms associated with œdema, especially such visceral manifestations as dyspnœa, are due to this latent œdema. The gross visible subcutaneous œdema he regards as much less dangerous, and only of importance because of the inconvenience it causes, and its liability to induce morbid conditions of the skin.

Widal also points out that the onset of pre-œdema can be readily detected by weighing the patient daily. Frequent estimations of the urinary chlorides are scarcely practicable to the majority of physicians. The permeability of the kidneys to salt may be determined by allowing more and more salt, until the weighing machine shows that pre-œdema is at hand. The amount of salt allowed is then reduced well below this limit; and the physician need trouble himself about it no further, unless another increase of weight gives cause to suppose that the renal permeability has been still further diminished.

In 1904, Olmer and Audibert published seven cases of ascites, due to cirrhosis of the liver, in which they obtained results very comparable with those of Widal in renal œdema. No one of these cases showed any sign or symptom of renal disease. In almost all there was considerable retention of chlorides. They tried the plan, so much followed in France, of forced excretion; and, by adding large doses of salt to the diet, they found that not only the natural but also the forced excretion of salt was diminished.

Unfortunately they limited these experiments to giving added salt and determining the amount of chloride retention, making few direct observations on the effect on the ascites, and in no case restricting the patient to a saltless diet. In one or two of their cases, the addition of salt caused paracentesis to be more frequently necessary. It is also noteworthy that the case, which presented little or no chloride retention, seemed to justify the good prognosis which they gave; as the ascites rapidly disappeared without any operative interference. Widal has also noticed the relation of chloride retention to the amount of ascites in hepatic cirrhosis, and he has found that saltless diet helps to diminish the fluid.

Similar results have been obtained in cases of ascites by Achard and Paisseau, Chauffard, and also Nobécourt and Vitry.

As a further instance of the relation between retained chlorides and serous effusions, the results of Achard and Laubry may be quoted. In cases of pleural effusion, they found that the administration of ten grammes of salt caused increase of the fluid, and that, broadly speaking, the amount of effusion varied inversely with the quantity of sodium chloride excreted in the urine.

Nobécourt and Vitry have also noticed retention of chloride in cases of tuberculous peritonitis with effusion.

In cardiac œdema, similar results have been obtained by Widal (working with Froin and Digne), and also by Vasquez and Laubry, Achard and Paisseau, and others.

Some of the results enumerated above are so striking that it is curious that they have received so little attention in this country. Some time ago, at the suggestion of Dr. Herringham, I made prolonged observations on three cases of ascites, and one case of parenchymatous nephritis, which were treated alternately on saltless and salt-containing diet. The results were interesting, but space does not permit them to be repeated here in detail, and they are to be found in the *St. Bartholomew's Hospital Reports* for 1905. In all cases we found that deprivation of salt seemed to exercise a beneficial effect, not so much in dispersing the fluid, as in delaying its accumulation. Even though in our hands the treatment proved far less successful than in France, still the slight advantage we found was quite sufficient to justify its use. Most cases to which it is applied are of a very chronic nature, and especially resistant to treatment. Any further means of relieving such patients is to be welcomed. If, with saltless diet, a case of ascites has to be tapped, say every three weeks, instead of every fortnight, a very real advantage is obtained.

Such being the case, one naturally turns to the consideration of the means by which the treatment may be supposed to diminish the œdema. If we turn first to the cases of nephritis, we find a difficulty arises at the outset, for, on reading through the numerous papers dealing with the relation of chlorides in the urine to the functional capacity of the kidneys, one is surprised by the contradictory nature of the results. There seem to be at least two distinct reasons for this. In the first place, it must be emphasised that, before any real knowledge of the chloride excretion of a patient can be obtained, careful daily determinations must be made for a considerable period. Unless this is done, we may easily fall into error; and it is not improbable that some of the authors to be quoted have not paid sufficient attention to this point. I have myself found that the chloride elimination of a patient, on a fixed diet, may show most marked daily variations.

Secondly, in these investigations, cases of various kinds of kidney disease have been used without much distinction as to their nature. Märishler, it is true, examined seven cases with this point in view. He found that, in the parenchymatous cases only, were chlorides retained; and he concluded that, in these, the retention was not due to impermeability of the kidney, but to a diminution of its water-secreting power.

Mohr, however, holds that, when uræmia is imminent, chlorides are diminished in the urine. Bohne goes so far as to think that retention means the onset of uræmia; while Strauss regards retention of chlorides as an index of renal inadequacy. Halpern seems to agree with the theory which we shall see is held by Widal; and the names of Noorden, Rosenstein, Fleisher, and Frerichs are also quoted by Mauté as supporting the view that the kidney is less permeable to chlorides in disease than in health.

On the other hand, many authors take a diametrically opposite view. Hoffman finds that, at the onset of uræmia, there may be an increased output of chlorides ; while on the other hand very marked retention may be present without the appearance of any disturbing symptom.

Among the many theories dealing with this subject, there are three that demand special attention.

According to one theory, the retention of chloride in kidney disease is due to the morbid condition of the kidney itself, a condition which leads to relative impermeability of the organ to chlorides.

The second theory maintains that the retention is a simple physical process, entirely secondary to the retention of water in the body.

The third theory is that, in these cases, the body tissues are in some way altered. so that they are enabled to hold, in combination with themselves, a larger amount of salt than usual.

The first two theories must be discussed more fully ; of the third there is less to be said. There is small chemical, or clinical evidence that the tissues are able to combine with large quantities of chloride ; but, at the same time, it is difficult to prove that they cannot retain more than usual in certain morbid conditions. The analogy of pneumonia, in which there is some evidence to show that part of the retained chlorides is held in the pneumonic exudation, is a slight presumption in its favour. We may certainly be prepared to admit this theory, as a partial explanation of the phenomena, although it is difficult to believe that it can account for them all.

THEORY OF RENAL IMPERMEABILITY. WIDAL'S THEORY OF CHLORÆMIA.

Widal maintains that the retention of chlorides in nephritis is due to what he describes as impermeability of the kidneys to sodium chloride. He holds that the secretion of salt is a highly specialised function of the kidney, and may be affected by disease quite independently of the other functions of that organ. He and Javal have published a case, in which the excretion of urea and phosphates was quite natural, while chlorides introduced by the mouth were largely retained. In fact, they say that chloride retention may co-exist with increased excretion of urea, as in another of their cases, in which loss of weight coincided with an increased chloride retention. They found that the amount of urea in the urine was excessive, and supposed that the loss of body weight was due to the extensive muscular catabolism which more than counterbalanced the increasing œdema.

Widal holds that this impermeability of the kidney to chlorides is never absolute, and that it varies largely from time to time, and in different cases. But he gives to his theory a very wide extension, holding that nearly all the distressing symptoms of nephritis are due, not so much to the œdema, or to the presence of unknown toxins, as to the saturation of the body with excess of sodium chloride. He and his school have published several papers with the object of showing how the various manifestations of kidney disease are due to salt. In fact to the greater part (if not the whole) of the symptom complex of nephritis, he gives the name chloræmia.

To give instances:

Javal found that the stools of patients, suffering from retention of chlorides, contained appreciably more salt than those of healthy persons. He was unable to cause diarrhœa by giving quantities of salt, but if the diarrhœa was already present, or was induced by improper feeding, it was observed that, on forced doses, the fæces contained an increased amount.

Widal and Javal examined cases of vomiting in nephritis. They state that the vomited matter contained a higher percentage of salt than usual, and also that saltless diet diminished the vomiting. They regard the symptom as an effort on the part of the body to get rid of salt. They have also contended that albuminuria is largely dependent on salt, and that deprivation of salt diminishes it.

Widal states that many of the nervous symptoms of nephritis are due to the action of excess of salt on the cortical and bulbar centres. Jacquet finds that saltless diet benefits the coryza, and Ravaut that it cures the skin eruptions in patients with albuminuria.

Lastly, Widal says : " La retention chlorurée est loin d'être la cause de tous les symptomes dits urémiques, mais elle est l'origine de beaucoup d'entre eux."

A theory of such far-reaching significance calls for careful criticism. First, as regards the papers just quoted. It is difficult to believe that, because the matter vomited by one of these patients is rather rich in salt, the vomiting is therefore necessarily an effort to get rid of the salt. It appears easier to think that the tissues of the patient were, at the time, as it were, saturated with œdema fluid, which contains about 6 per cent. of salt, and that some of this very naturally passed into the vomit. I have examined the figures in these cases, and have found that the percentage of salt in the vomit (or fæces) is lower (and usually very much lower) than the 6 per cent. which we know to be present throughout the body.

Let us now turn to the question of the supposed power of the diseased kidney to keep back salt. It must be admitted at once that, on any theory of renal secretion, it is not easy to explain such a retention. I know of no direct experimental evidence that the diseased kidney cannot secrete salt ; while the two following observations afford very valuable testimony to the opposite possibility.

Teissier and Paul Courmont have recorded a most interesting case of what is best described as a combination of chronic nephritis and diabetes insipidus. The patient exhibited all the essential symptoms of diabetes except glycosuria. In addition, there were albuminuria and other evidence of nephritis, but no œdema. The distinctive feature of the polyuria was the excessive amount of salt passed daily. They thought this due partly to increased renal permeability, and partly to inability of the tissues to hold salt ; because, on forced doses, the hyperchloruria was increased out of proportion to the salt taken. Whatever the explanation of the case, it is quite certain that the kidneys were diseased, and that an excess of salt was excreted in the urine. Thus, in a diseased kidney, the renal permeability was increased rather than diminished.

The second case bearing out the same view is that published by Lépine and Aubert. They found that, in an animal with one kidney experimentally injured, the injured kidney secreted more chlorides than the normal one.

Again, the effect of such chloride retention, acting over a long period, must not be forgotten.

Such patients often remain in much the same general condition for weeks and months ; and, if they were accumulating in their bodies so many grammes of salt daily, the total amount would soon become enormous, and one might almost imagine the patient crystallising out as a second Lot's wife. As a matter of fact, we know that no such accumulation of salt takes place, that the percentage of salt in the body fluids remains remarkably constant. There is no evidence that any large amount is stored up in the tissues. Widal, indeed, has laid stress on the way in which such a patient varies in his power to excrete salt. He looks upon this as affording an argument to show that the retention is essentially renal, and assumes that more chloride is passed, because the renal cells have temporarily recovered their secretory power. As we shall see, the supposition that retention of chlorides is secondary to retention of water affords a much simpler explanation.

An essential part of the theory of chloræmia is that the excess of salt in the body exercises a deleterious effect on the tissues. With the exception of the statement that the body is always striving to get rid of the salt, no evidence of this is forthcoming. On the other hand, certain experiments, carried out by Castaigne and Rathery, and by Lesné and Richet, tend to the supposition that such an amount of salt in the body is beneficial rather than the reverse. These experiments are, however, in both cases, somewhat inconclusive.

Again, if this doctrine of chloræmia is to be accepted, one naturally asks why the symptoms ascribed to it are not confined to the cases of parenchymatous nephritis with œdema and retention of salt ? They may all, or nearly all, be found in cases of interstitial nephritis, in which no retention of chlorides has been demonstrated.

THEORY THAT SALT RETENTION IS SECONDARY TO RETENTION OF WATER.

The second theory may be associated with the names of Marishler, Scheel, Limbeck and Dreser, Achard and Paisseau, Lépine and Aubert.

According to their theory, the presence of œdema is assumed; and the retention of salt is explained as being largely, if not entirely, due to the necessity of maintaining the body fluids at a definite osmotic pressure. It does not in any way exclude the possibility that a certain amount of the salt is held in some intimate connection with the tissues. Indeed, the belief that some salt may be thus accounted for may be a help towards the acceptance of the theory.

It is essential to the enunciation of this physical explanation that the presence of œdema is assumed. It would be out of place here to discuss the still open question of the cause of dropsy, whether renal, cardiac, or other. Quite recently Bainbridge has brought forward the view that the œdema of nephritis may be due to the increased quantity of catabolic products retained in the body. Bradford, in 1892, showed that, if more than a certain amount of kidney substance is experimentally removed, there is a very large increase in muscular metabolism. As Bainbridge has suggested, the osmotic power of the products of this increased metabolism, caused by the functional absence of healthy kidney tissue, may be a very important factor in the production of renal œdema. The causation of cardiac œdema also offers many difficulties to any complete explanation; and even that of the ascites, following on hepatic cirrhosis, is not quite clear.

If the fluid causing œdema, or ascites, is withdrawn and examined, it is found to contain a remarkably constant percentage of salt, the figures usually varying between 6 per cent. and 7 per cent. The amount of fluid thus retained in the body is frequently very considerable; and, accordingly, in order to maintain this percentage of sodium chloride, a relatively large amount must be made use of.

If the patient, with a tendency to œdema, is on an unrestricted diet, there will be no difficulty in obtaining this salt, as without doubt one habitually consumes far more sodium chloride than is required for the needs of the body. If at any time the œdema is increasing, more salt will be needed, and obviously less salt will be excreted than is ingested; in other words, there will be retention of salt. As the increased tendency to œdema is probably due to a deterioration in the condition of the patient, the increased retention of salt will coincide with a less satisfactory state of health. If, on the other hand, whether owing to treatment, or to any other cause, the tendency to œdema is diminishing, the reverse will take place. As Widal says, the retention of salt is never absolute. Some will be passed out of the blood by the kidneys. The blood we know has a most remarkable power of keeping its percentage composition within very narrow limits. Therefore, if salt escapes from the kidneys, it will be withdrawn from the tissue fluids. This process also necessitates the withdrawal of a certain amount of water, which will also escape by the kidneys. Were this not the case the tissue fluids would become hypotonic. In this manner, the diminution of œdema must be accompanied by an increased excretion of water, and what Widal calls a diminished retention of salt. Incidentally, owing to the prime cause of amelioration, the patient will almost certainly be better; and thus it is clear how the general condition of the patient may, *cæteris paribus*, vary inversely with the amount of retention of salt.

The same reasoning may be applied to the case of the patient who is given saltless diet. Supposing the œdema to be increasing, either the necessary salt would be obtained from the small amount which cannot well be excluded from the food, or the amount required might be withdrawn from that already present in various combinations throughout the body. In the latter case, the metabolism might be injuriously affected; and I am inclined to believe that in this lies the explanation of the few cases, that are reported, of patients, who cannot receive saltless diet without injury.

On the other hand, let us take the case in which the œdema is not increasing. We have already mentioned that the blood maintains its percentage composition with little variation. Even on saltless diet, Langlois and Richet have found that the amount of salt in the blood is not varied. Consequently, the urine will still contain a small amount of salt, and the blood will have to withdraw a corresponding amount from the body fluids, in order to make up for the deficiency so produced. As in the case stated above, water will necessarily pass out too. In the case of ascites, where the cause is, in part at least, mechanical, these processes may not be able to produce the absorption of as much fluid as is exuded; but they should obviously tend to diminish it, or, as in my cases, to delay its accumulation.

If we accept this physical theory as accounting for the retention of salt in these cases, it should theoretically be possible to discover an accurate numerical relation between the amount of salt retained and the amount of water constituting the œdema. It must be admitted at once that such a relation is hard to establish.

Olmer and Audibert, in one of their cases of cirrhosis, carefully estimated the chloride intake and output during the accumulation of the intraperitoneal fluid. In this way, they knew how much salt had been retained. On examining the ascitic fluid, obtained by paracentesis at the end of the period, they were unable to recover as much salt as had been retained in the body. They point out, however, that they doubtless failed to empty the peritoneum at their operation. As a further criticism, I would suggest that possibly the patient's legs, if not actually œdematous, were in a stage of pre-œdema, and so might retain a considerable amount of fluid.

M. R. Marie conducted similar experiments in cases of cardiac and renal œdema. Estimating the amount of fluid lost on a saltless diet by the diminution in the patient's weight, he compared it with the excretion of salt, and found that the two figures were very far from corresponding. For instance: during treatment, a man lost 7·8 kilogrammes in weight; and this, Marie assumed, represented that weight of water. If this fluid contained 6 per cent. of sodium chloride, he should have excreted 46·80 grammes of salt during the same period. As a matter of fact, he excreted 158 grammes. To put it from another point of view, he excreted enough salt to account for a loss of weight of 26 kilogrammes.

One would like to know whether the estimation of the salt was carried on over a sufficiently long period; and secondly, whether, owing to improved conditions, the patient was not putting on weight as flesh, while losing it as water.

The results of both these experiments need confirmation, especially in view of the fact that, in the December number of THE PRACTITIONER, Bainbridge published far more successful results. In his case, the theoretical retention of water was 4·5 kilos, while the actual increase in the patient's weight was

4·7 kilos. A more accurate correspondence than this could hardly be expected.

It is interesting to consider the two main theories of the mechanism of renal secretion, with the view of seeing how much light either the one theory or the other can throw on the problem of salt retention. According to the Bowman-Heidenhein theory, the kidney secretes urine by a vital secretory act. The cells of the glomeruli secrete water, and the saline constituents of the urine, including sodium chloride. The cells of the tubules secrete urea, uric acid, the other specific urinary constituents, and a little water. If we try, on this theory, to explain retention of salt in nephritis, we have to assume that the glomerular cells are so altered by disease that they not only fail to secrete salt, but also prevent its passage through them by any physical process. Salt is one of the most diffusible substances in the body ; and we know that damage to cells almost invariably makes them more rather than less permeable. If we are prepared to accept the Bowman-Heidenhein theory, we must admit that it is improbable that the damaged kidney can retain salt.

According to the followers of Ludwig, the renal glomeruli act as filters, and allow the passage of a fluid which, so far as its diffusible constituents are concerned, is identical with the plasma. During the passage of this glomerular filtrate down the tubules, it becomes concentrated by giving up some of its water and salts to the surrounding lymph. Now this concentration of the urine has been calculated to require the expenditure of a very large amount of energy ; and it is the explanation of the source of this energy which presents so many difficulties. The possibility of such a concentration has been rendered much more acceptable by the recent researches of Cushny. He has made very numerous and laborious experiments, and obtained results which tend to show that the layer of living cells lining the tubules acts as a semi-permeable membrane, which allows of the passage through it of salts and water, but which is almost impermeable to urea, sulphates, and phosphates. In his first series of experiments, he induced saline diuresis by means of the simultaneous injection of sodium chloride and sodium sulphate. He found that, while a rapid diuresis was in progress, a large quantity of chloride was excreted, but that, as soon as the flow became slower, the chloride almost disappeared from the urine, while the excretion of sulphate continued. He explains these results by supposing that, during the height of the diuresis, the flow through the tubules is so rapid that there is little time for any re-absorption to take place, and so the urine secreted resembles very closely in composition the true glomerular secretion. This explanation was confirmed by a second series of experiments, in which he induced the diuresis in the same manner, but delayed the flow through the tubules by means of slight pressure on the ureter. By this means he found he was able to bring about the re-absorption of almost the whole of the chlorides.

If we now attempt the explanation of chloride retention, according to the Ludwig theory, we still meet with difficulty. Assuming, as the theory assumes, that the secretion of glomerular urine is essentially an act of filtration, we can hardly suppose that damaging the filter would render it less permeable to any constituent, and certainly not to one such as sodium chloride. Turning to the tubules, we again cannot suppose that their cellular lining becomes more permeable to salt, for the theory assumes that it is permeable to begin with.

There is, however, another possible explanation. Cushny has shown that a sluggish flow through the tubules increases the amount of chloride absorbed, and decreases that in the urine. Now, in the case of parenchymatous nephritis, and the cases described are always of this nature, the amount of urine is scanty, and the flow is correspondingly slow. We have then present, in such cases, all the conditions shown to be favourable to a large reabsorption of chlorides. Also we have here a reasonable explanation of the great variations in the daily chloride excretion of such cases; for we have only to assume a very possible daily variation in the rate of flow (not necessarily the amount) due to amount of fluid taken, drugs, temperature, or nervous conditions. We have then here a possible renal causation of the diminution of chlorides in the urine, just as the case, already quoted, of hyperchloruria and nephritis gives an excellent example of the opposite condition. Such a cause can hardly be justly described as renal impermeability, as it depends on an increase rather than a diminution of this function of the kidney, and is entirely secondary to the lessened flow of water. We have here also an explanation which is applicable, not only to renal cases, but just as fully to patients suffering from œdema of hepatic, or cardiac origin ; as, in both these conditions, the amount of urine is usually much diminished.

In support of this point, and also of the general contention that retention of salt is secondary to that of water, may be mentioned the following observation of Huchard. He found that the diuresis of digitalis was accompanied by increase in the urinary chlorides, and this was confirmed by Merklen, who noticed that this hyperchloruria ceases when all the œdema has disappeared.

This influence of the slowing of the stream of urine down the tubules also furnishes a possible explanation of other cases, in which retention of chlorides has been described. Of these, the most numerous are the various conditions associated with high fever. In all of these, the diminution of the urine is a marked symptom. Retention has also been recorded in diseases, such as tuberculous peritonitis and appendicitis, and in these also a similar explanation is applicable. In conditions such as pneumonia and malaria, in which the retention is so very marked, other and additional causes are probably at work.

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MODERN LEADERS IN MEDICINE.

I.

SIR WILLIAM JENNER.

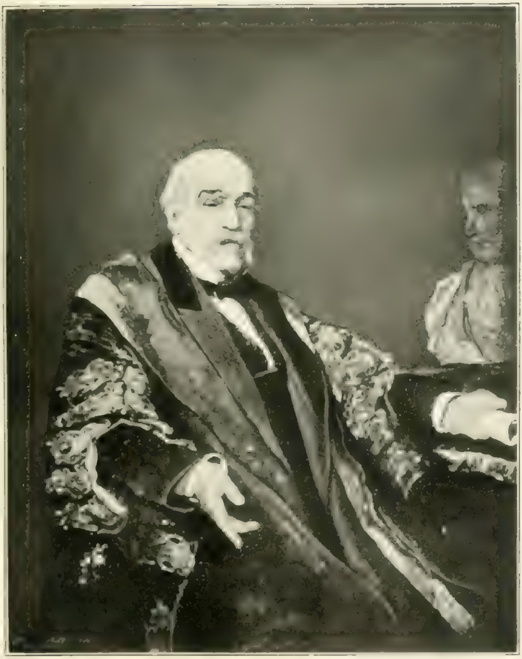
[With Plate XIX.]

WILLIAM JENNER was for many years the acknowledged head of the medical profession of this country, and the foremost member of the staff of University College Hospital at the time of its greatest renown. For several years he ruled the London College of Physicians with a strong hand. He was for a long time the trusted counsellor of a Sovereign, whom long experience had made an unusually good judge of men, and she had the insight to detect the sterling worth of the man beneath the blunt manner. As a physician he was great because he set before himself the highest standard of professional efficiency, and concentrated the whole force of a massive intellect and dominating will upon the case before him. Though a man of masterful personality, he was of the strictest intellectual honesty, and, unlike many of the Olympians of Medicine, scorned to hide a mistake or throw the blame on another, and would take a suggestion from a clinical clerk or even a nurse, if he thought it sound.

Of somewhat lowly origin, he was born at Chatham in 1815, and studied medicine at University College, London. He passed the "College and Hall" in 1837, and so doubtful were his prospects at that time that, as he once said in an address to students, he had to take a position which the humblest of the humble among them would have despised. The legend is that he kept a small "doctor's shop" in Albany Street. He was able to continue his studies, however, and in 1844 he became a Doctor of Medicine of the University of London. He was admitted a Member of the Royal College of Physicians in 1848, and was elected a Fellow in 1852.

It was in 1847 that Jenner began, in the London Fever Hospital, a systematic investigation which led to the final establishment of the doctrine that typhoid fever and typhus are distinct diseases. His observations, which were conducted with conscientious care, were made on a series of 1,000 cases. The result was, to use his own words, to prove incontestably,

PLATE XIX.



SIR WILLIAM JENNER, BART.

so far as induction can prove the point, that the specific causes of typhus and typhoid fevers are absolutely different from each other, and to render it in the highest degree probable that the specific cause of relapsing fever is different from that of either of the two former. Typhoid fever had already been clearly differentiated from typhus by Louis in France, and by A. P. Stewart in this country. What Jenner did was to furnish conclusive demonstration of the truth of this doctrine, and to force conviction on the mind of the profession.

In 1849, he was appointed to the chair of pathological anatomy in University College, London, and soon afterwards he became assistant physician to the Hospital. Five years later came promotion to the full physicianship, an office which he held for twenty-two years. In 1860, he succeeded Edmund Alexander Parkes, the famous army hygienist, as Holme Professor of Clinical Medicine. From 1863 to 1872, he occupied the chair of Principles and Practice of Medicine. For eighteen years he was physician to the London Fever Hospital, and for ten years to the Hospital for Sick Children in Great Ormond Street. In these various fields of observation, he gathered the stores of clinical knowledge for which he was almost unrivalled among his contemporaries. He was, therefore, fully prepared, when there came the tide in the affairs of men that leads on to fortune, to take it at the flood.

In 1861, William Baly, who had been appointed Physician Extraordinary to Queen Victoria in the previous year, was summoned to Osborne. He reached the station just in time to see the train start. On his hastily explaining the circumstances, it was stopped, and he got in. An accident occurred and the unfortunate physician was killed, being the only passenger who suffered that fate. Jenner was appointed to the place thus, as it might seem, providentially made for him. Almost immediately he had an emergency to deal with that tried his mettle to the utmost. The Prince Consort fell ill with typhoid fever, and the responsibility of his treatment fell wholly on Jenner. He proved himself equal to the burden laid upon him, and although it was not his good fortune to save the precious life entrusted to his charge, he gained the confidence of the bereaved Queen so fully that, in 1862, she made him her physician in ordinary.

In the following year, he was appointed physician in ordinary to the Prince of Wales, whom, in 1871, he helped to save from the jaws of the same death which had carried off the Prince Consort ten years before.

Jenner enjoyed the confidence of the whole Royal Family. In his attendance on the Queen at Windsor and elsewhere he was a privileged guest, being allowed, contrary to the usage of the Court at that time, to take his own valet. Though devoted to his Sovereign, he never sacrificed his independence to his loyalty. His speech was too plain not to be offensive sometimes to royal ears, but the position, which he held in the regard of those whom he served so well, was too strong to be shaken. On a memorable occasion the Queen's interest in a dying statesman led her to ask Jenner to do what he deemed would entail a sacrifice of professional principle. He refused, but on the request being pressed, he is said to have replied that, as Her Majesty's servant, he was bound to obey her command, but it should be for the last time. A way of escape from this awkward situation was found in a compromise, and though the independent physician was condemned by the voice of the public, he was applauded by his own profession.

Jenner's services to the Court were rewarded with exceptional distinctions. He was created a baronet in 1868, and a Knight Commander of the Bath in 1872; the Grand Cross of the Bath was conferred upon him in 1889. He was also a Commander of the Order of Leopold of Belgium. From his profession he received all the honours in its power to bestow. He held all the offices of the College of Physicians, being Councillor in 1865–6–7, Censor in 1870–1 and in 1880, Harveian Orator in 1876, and President from March 1881 to March 1888. He was also at different times President of the Epidemiological Society, of the Pathological Society of London, and of the Clinical Society. The Royal Society gave him the blue ribbon of science by electing him a Fellow in 1864; Oxford, Cambridge, and Edinburgh gave him honorary degrees.

In 1890, Jenner, feeling that his strength was breaking down under the stress of a laborious life, gave up the practice of his profession and spent the remaining years of his life in retirement. He died at Greenwood, near Bishops Waltham, in Hampshire, in 1898. He left a large fortune which, however, was not wholly gained by professional work. He married Miss Adela Lucy Leman in 1858, and had a family of five sons and one daughter. The only one of his sons who followed his profession was Louis, a pathologist of great promise, who died not long ago in the early prime of life.

Jenner wrote little, but his contributions to medical literature were of enduring value. The papers on the distinction between typhoid fever and typhus, which laid the foundation of his fame, appeared in the *Monthly Journal of Medical Science* for 1840, and in the *Transactions of the Royal Medical and Chirurgical Society* (1850). Among his other writings are a treatise *On the Identity and Non-Identity of Typhoid Fever*, which saw the light in 1850; a short monograph on *Diphtheria, its Symptoms and Treatment*, which appeared in 1861; a collection of lectures and essays on Fevers and Diphtheria, 1849-79, published in 1893, and *Clinical Lectures and Essays on Rickets, Tuberculosis, Abdominal Tumours, and other Subjects*, published in 1895. But much of the best of Jenner's teaching is to be found only in the notebooks or memories of his pupils. As a teacher of the fundamental truths of medicine he was unsurpassed. Though of an impetuous temper, that made him the terror of officials of every degree, his patience with a dull student was in strong contrast with the conduct of those superior persons who are always glad to seize such a chance of giving a display of wit. Without any pretension to brilliancy of style or epigrammatic felicity, Jenner had the gift of concentrating the essence of his priceless experience in a telling phrase, often set in an apt illustration, that impressed indelibly on the memory the truth which he wished to convey. So deep indeed was the impression that the recipient sometimes came to look upon it as his own, and—doubtless in all innocence—published it afterwards as an original addition to the sum of knowledge.

The extent to which Jenner's teaching infiltrated the minds of the men, who came under its influence, can be adequately estimated only by those who know what the art of healing was in his early days. There was then a remedy for every disease, and it was mostly of the drastic kind. Fevers were "cut short"—and with them too often the patient—and where no supposed "specific" was available, the practitioner fell back on the "good old rule, the simple plan" embodied in the *seignare* and *purgare* of Molière's candidate. Jenner was one of the pioneers of reform. He insisted on the truth that acute fevers runs a definite course, and that "we have no drug to cut short the fever, no drug that exercises, so far as is known, any influence on the specific disease." For this he was called a sceptic in medicine, a charge which he vehemently repelled. An excellent illustration of his therapeutic attitude is given by himself in an address delivered before the British Medical Association, and reprinted in a booklet, entitled *The Practical Medicine of To-day* (1869). The passage is worth quoting, and it may serve as a specimen of his virile manner of speech, as well as of his teaching :—

"I was one of three who met in consultation concerning a case of apoplexy. In the opinion of one of my colleagues and myself, the only treatment to be adopted was as follows :—To place the patient in the recumbent position, with head and shoulders raised ; to enforce absolute rest ; to keep the bowels so far loose as to prevent excitement and straining ; to apply cooling substances to the head in the event of any heat of the part occurring ; to support the patient with light nutritive food, having regard to his habits. The third gentleman protested against the modern system of doing nothing ; he was anxious to bleed, to purge, to blister ; and, when opposed, was not sparing of the word 'sceptic,' etc.

"Now the difference of opinion in this case was not due to scepticism on the one side and justifiable faith, *i.e.*, faith justified by knowledge, on the other, but to knowledge on the one side, and absence of knowledge on the other.

"The case was one of degenerative change, retrograde metamorphosis, of the arteries : one had become so rotten that its wall had given way, its contents had escaped, a clot had formed, and by its mechanical effects had given rise to the symptoms. The heart shared in the degenerative changes, the bleeding had ceased. To those who understood the real nature of the case, the lesions present, and the mode in which they had been produced—in short, the pathology of the case—belief in the efficacy of so-called active treatment appeared to be not merely unjustifiable faith, foundationless faith, faith without knowledge, but to be faith in opposition to knowledge, which in medicine is the worst form of scepticism, inasmuch as it is doubt of truth and belief in error—doubt which may prevent the saving of life, and belief which, embodied in practice, may kill."

From an address to the Epidemiological Society in 1866, reprinted in the same booklet, it may be gathered that Jenner was an early advocate of views on the teaching of the laws of health in schools and the training of the mothers of future generations which are scarcely yet regarded by legislators as within the sphere of practical politics. Speaking of rickets, he said:—

"The causes of rickets are, poorness of the mother's blood, errors in diet, *i.e.*, feeding the child with food unsuited to its wants and its digestive powers—and, as subsidiary causes, deficient light and impure air, produced especially by overcrowding of the sleeping room. Poverty—inevitable poverty—plays a great part in the production of some of these causes. If society did its duty in providing suitable abodes for the poor, they would suffer little from want of light or overcrowding at night. The anæmia of the mother would be less, and her blood better fitted to nourish the infant. Ignorance of the proper mode of feeding the child assists in the production of rickets in a larger degree than poverty. Judging from my own experience, I should say that rickets, so severe as to lead even indirectly to death, would be comparatively rare, did the poor know how to feed a young child—were the poor aware of the necessity of the infant being fed with food fitted to its age. Law can do something here; for it can make compulsory the teaching of *practical* laws of health in all schools supported in any degree by the public money. To teach young girls how not to destroy their future children, is surely as important as to teach them much of what is now considered essential for them to know. I would have an infant nursery attached to every national girls' school, so that the girls might be practically taught how to fulfil their practical duties to their family and to society. Diffusion of practical knowledge is the great preventive remedy of rickets. Law can aid in the spread of that knowledge, and society, if it did its duty, would remove the subsidiary causes of want of light and overcrowding."

Jenner's character as a clinician was marked by the rugged honesty that distinguished him as a man. He could not have stooped to astonish his pupils by making an apparently instantaneous diagnosis of the case of a patient at the farthest end of a ward, without thinking it necessary to inform them that he had taken the Napoleonic precaution of preparing his *coup de théâtre* beforehand. Not from his lips could have come the euphemisms and evasive paraphrases which won popularity for more diplomatic rivals. Where the outlook seemed to be desperate, he could not prophesy smooth things. It is related that he was called to a case of cancer of the liver, in which there could be no comforting shadow of doubt as to the nature of the disease. Jenner told the friends the bitter truth, adding that he could do nothing for the patient. Another physician was called in, who said, although there certainly was "a swelling probably not altogether benign in character," life could be prolonged, and the end made easier. The one got his single fee, whereas the other pocketed a substantial sum for a daily attendance of several months. The story is characteristic of Jenner, but there is a good deal to be said for his suaver colleague.

In Jenner's professional, as in his personal, character there was much of the typical John Bull, whom, it may be added, he was not unlike in appearance. If his intellect was strong, so were his prejudices. He refused to accept the discovery, made independently by Waller and Cohnheim, of diapedesis, saying that he would as soon believe that a brick could pass through the wall of a house as a white corpuscle escape through the wall of a blood vessel. Long after Meyer's discovery of adenoid vegetations, Jenner plainly hinted that they were inventions of the specialist imagination. In a debate as to the admission of women to technical degrees, he declared that, dearly as he loved his only daughter, he would rather see her in her coffin than working in a dissecting room.

With all his superficial asperities, however, he was a man of the most genuine kindliness of heart, and of unstinted generosity to those in need. In an age of advertisement he hated the "public ways which public manners breeds," and consistently shaped his conduct in accordance with the noblest traditions of the profession, in which he was so long the most conspicuous figure.

NOTES FROM FOREIGN JOURNALS.

SENATOR ON ARTERIO-SCLEROSIS.

The treatment of this disease is a matter of recent date, although the changes in the arteries, which constitute arterio-sclerosis, were fully described by Bizot, Rokitansky, and Virchow. Inflammation of arteries was talked about, but seemed hardly accessible to diagnosis, and consequently much less to treatment. As regards the causes of arterio-sclerosis, it is now known, mainly upon the authority of Traube, Senhouse, Kirkes, and Huchard, that the disease arises from constant increase of the blood-pressure in the arteries. Increased blood-pressure is one of the symptoms accompanying chronic nephritis, a disease which, in time, leads to contracted kidney and arterio-sclerosis. Experiments have recently been carried out in order to ascertain the effect produced on arteries by increase of blood-pressure, artificially induced by injections of adrenaline or renoform. Certain changes, bearing some likeness to arterio-sclerosis, were found, in the vessels, when this artificial pressure had been kept up for some time; but, at the same time, it was found that the pressure-raisers produce these changes in the presence of bodies whose action prevents the rise of pressure, such as iodide of potash and nitrite of amyl. Thus it is very doubtful whether the cause of the arterial changes is not a special toxic effect of the agent injected rather than the rise in pressure. In fully-developed arterio-sclerosis, we find hyperplastic inflammatory processes mingled with degenerative, but it is still a moot point whether the change begins in the intima or in the muscularis. Most likely the point of outbreak differs according to the vessel-region, and is not the same in the aorta as in the small arteries; in the former the process, as Virchow has pointed out, certainly starts in the intima as "endarteritis chronica deformans."

It is beyond question that the calcareous deposit is a secondary process in every case; that is, the lime is deposited on already-diseased coats. Hence decalcification, far from leaving the vessels healthy, will, on the contrary, leave them worse off, for they will be softer and less capable of resistance, predisposing the decalcified vessel to rupture sooner perhaps than the calcified. The decalcifying treatment, which has recently been recommended for arterio-sclerosis as specific, must for this reason be condemned.

The statement, that arterial pressure is raised in developed arterio-sclerosis, depends upon the theoretical conception that this rise is the cause of the sclerosis, which, as has been stated already, is not borne out by experiment. Moreover, the estimation of the blood-pressure in this condition of the vessel is rendered very difficult by the resistance due to the thickened coats of the vessel. Clinically, we know that arterio-sclerosis may be said to be a physiological phenomenon of old age; but we cannot very well suppose that, with advancing years, when the organs are getting worn out by use and weaker, the blood-pressure increases, and, in that way, originates the sclerotic condition.

Among the proved ætiological factors are chronic intoxications and infections, endogenous and exogenous, such as chronic syphilis, old-standing abuse of tobacco, and chronic metallic intoxication—lead, mercury, and, it seems, bi-sulphide of carbon. Senator is inclined to ascribe a direct influence to abuse of food-accessories, such as strong coffee and tea, meat extracts and smoked foods ; an auto-intoxication taking place just as in the endogenous diseases, gout, diabetes, and chronic nephritis. In the case of the last-mentioned, H. Straus has proved that nitrogenous bodies, which are not albumins, accumulate in the body. He (Senator) is also of opinion that the disease is encouraged by a too-plentiful use of nitrogenous food, since it is not at all infrequently met with in athletes, who avoid carbohydrates and fats in order to escape putting on flesh, and who take proportionately more meat, wurst, and cheese. He also suggests that the present custom of eating plenty of meat, especially at breakfast, is one of the reasons for the prevalence of arterio-sclerosis.

Albuminous food gives rise to intestinal decomposition-products in much larger amount than is the case with vegetable food ; one can, therefore, readily imagine a chronic intoxication by absorption of these products, through which arterio-sclerosis is produced. The disease is common among those who lead a sedentary life, and get very fat, especially in fat-bellied women at the time of the climacteric. In these cases, decomposition-products are formed in the gut, in consequence of the want of exercise, and of the constipation almost invariably present, which is, moreover, promoted by the abundance of fat in the abdominal walls, the omentum, and mesentery. It is also possible that this excessive deposition of fat renders the passage of the blood through the vessels more difficult, giving rise to increased pressure, which may share in causation.

As regards treatment, the prevention of such causes, as are known, must always be borne in mind, and carried out so far as possible. Senile arterio-sclerosis cannot be arrested, but its premature onset may be warded off by removing, or preventing, everything which, in early years, favours the development of sclerosis, as well as by treating all diseases from which it tends to arise. The patient must be removed from all chance of exogenous intoxications (lead, etc.), and auto-intoxication, by misuse of food-accessories, must be checked. In carrying out the latter, due regard must be paid to the individual and his habits, for a sudden deprivation and violent change of the whole mode of life has the worst possible results. The alteration must be made gradually.

It is most important that the patient should be suitably nourished, and it is best to exclude, so far as possible, all nitrogenous food-stuffs like meat, especially the very albuminous. A diet of milk and vegetables is most suitable, in which carbohydrates and fats predominate. Albumin is only present in the absolutely necessary amount, and extractives are altogether absent. Such a diet will include milk, bread, farinaceous foods, porridge, fats, fruit, vegetables, eggs in small amount, and, if any meat, then the so-called white meats and fish, which are relatively poor in albumin. In the case of stout people, some modification is obviously necessary as regards the carbohydrates. Sugar and starches must be eliminated, and green vegetables, fruit, and lean meat given, the bowels must be kept well opened, and muscular exercise undertaken in moderation, and with due care of the heart's action. The lacto-vegetarian diet not only lessens auto-intoxication, by reason of the reduction in the products of intestinal decomposition, but recent researches have shown that the viscosity of the blood is lowered under its influence. The work thrown on the heart and vessels is therefore eased to their great advantage.

It is to the last-mentioned result that the good effect of iodine preparations is apparently due, and it is possible that they exert some further good effect upon the coats of the arteries, especially upon the intima. Their action is increased by combining with them remedies which act as vasodilators, like the nitrites. Senator has long made use of this combination :—

℞. Potass. (vel Sodii) Iodid. - - - - gr. vj.–viij.
Sodii Nitrit. - - - - - - gr. j.–ij.
Aq. - - - - ad ℥ss.
M. ter quotidie sumend.

Or this may be preferred :—

℞. Tinct. Iodi. - - - - - - ♏ xvj.
Sp. Æther. Nitrosi - - - - ℥j.
M. Fiat. Mist.

"Take 20–30 drops three or four times a day."

A course of hot baths, or even simple warm baths, will promote the same object, viz., dilatation of as large a part as possible of the arterial distribution, and lessening of the blood-pressure. If, however, there is extensive disease of the vessels, and the heart is implicated, these are better replaced by CO_2, or even oxygen ("Ozet") baths. Some cases will benefit by friction and douches, others by massage and active and passive gymnastics, but each case must be dealt with individually with careful regard to the heart. Finally, must be mentioned a method, which has been much praised of late, based upon the idea that in arterio-sclerosis there is an abnormally small amount of salts in the blood. Consequently, a mixture of the salt constituents of the blood is administered, and this is obtainable, either as Trunecek's serum, or as "Antisclerosin." The fact as to common salt has been proved by Dennstedt, Rumpf, and L. Weil, and, according to the last-named, the lime contents of the blood are reduced, whilst the arteries of the kidneys are abnormally rich in lime. It is, of course, questionable whether this is cause or effect, but, in any case, the poorness in salts is an abnormality, so that the prudent introduction of blood-salts can do no harm, and may even be useful, provided that they can be made use of and not excreted unused. Of the three, v. Poehl's "Sal. physiologicum," Trunecek's serum, and "Antisclerosin" tablets, the last is most suitable for use. Sodium chloride is the chief constituent, then sodium sulphate, sodium phosphate, sodium carbonate, magnesium phosphate, and calcium glycerophosphate: all in the same proportion as in the blood. One begins with two tablets a day, and gradually increases to six a day. Senator thinks they do good, if taken with due regard to digestion, but, as he has always given them in addition to the iodine and nitrite combinations, and also with the dietetic restrictions mentioned, he is not able definitely to decide whether they have any specific action or not.

ADRENALIN IN OSTEO-MALACIA.

Bossi reports, in the *Policlinico*, a remarkable case of cure. A woman, aged 38 years, was admitted under him, in the eighth month of her eighth pregnancy.

The bones of the pelvis, the clavicles, and the ribs were softened and painful to the touch, and the pubis was already becoming duck-billed. The patient had been unable to walk for some months, and had remained in bed, but, in spite of nourishing food and tonic medicines, her condition continued to get worse. Bossi thereupon determined to try the effect of hypodermic injections of adrenalin. He was induced to do this by reason of its known vaso-constrictor properties, which might act upon the utero-ovarian circulation, by the favourable results which Stoeltzner is said to have obtained by supra-renal opotherapy in rickets, and, finally, by the fact that removal of the supra-renal bodies in animals produces marked changes in the ovaries. Accordingly, the patient was given an injection every day of half a cubic centimetre of adrenalin solution (1 per 1,000). From the time of the first injection improvement was obtained in the pain and insomnia, and the effect was increased after each fresh injection. Seven injections in all were given, after which the patient was able to walk, and an examination of the pelvis showed the absence of all pain in the bones, which, moreover, were recovering their normal consistency and shape. The patient went to full time, and had a normal labour, although, when first admitted, Cæsarian section was looked upon as inevitable. Bossi has great hopes that this wonderfully quick recovery has put him on the track of discovering the real remedy, and also the true cause of this affection, but he does not disguise from himself the fact that "one swallow does not make a summer."

ACUTE SUPRA-RENAL INSUFFICIENCY.

The supra-renal bodies may be involved in different affections, such as tuberculosis, hæmorrhage, inflammation, and growths, and these lesions are usually associated with pericapsular affections, and particularly with those of the great sympathetic system. Then we see a collection of symptoms, described as "Addison's disease." When, on the other hand, the lesions are limited to the capsules, morbid symptoms are produced, which are the result of supra-renal insufficiency, a condition which is seen under three forms, chronic, acute, and fulminating. The last two are the most frequent, and, according to Sergent and Léon Bernard, are characterised by a sudden onset, which suggests poisoning, an attack of cholera, atypical typhoid, peritonitis, septicæmia, appendicitis, or of salpingitis. These symptoms, in some cases, may be preceded by a period of fatigue and general lassitude. Melanodermia is not present. Asthenia is not noticed on account of the rapid course of the disease, but the following symptoms have been observed:--Lumbar pains, uncontrollable vomiting with or without diarrhœa, viscid sweatings, coldness of the extremities, sub-normal temperature, feeble pulse, and arterial hypotension. A fatal issue arrives quickly and suddenly. The affection attacks, more particularly, persons between the ages of twenty and thirty years. Treatment is difficult, but, if one has any suspicion of the presence of this disease, it is necessary to order complete rest, hygienic measures, tonics, glycero-phosphates, lecithin, and especially supra-renal opotherapy, which may effect a temporary improvement, or even a cure. *Journal de Méd. et de Chir. Prat.*

SALT-FREE DIET IN THE TREATMENT OF SCARLATINA AND OF ACUTE NEPHRITIS.

Delearde, in the *Echo médical du Nord*, describes the treatment and insists upon the *régime*. From the first is instituted as rigorous an antiseptic treatment as possible of the skin, the throat, and the nasal fossæ While, from the onset of the disease, the patient is fed during the whole eruptive stage by a diet from which all salt is omitted. He is kept absolutely quiet in bed, in a room maintained at a moderate temperature. Every day he is given a hot bath (98·6° F.), and washed with soap, to ensure the cleanliness of the skin, and to remove scales. After the bath, the whole body is, if possible, well rubbed with Eau de Cologne. In the case of a mild throat attack, the pharynx is brushed over three times a day with: —

℞	Resorcin.	gr. xxx.
	Glycerin.	℥j.

In more serious states of the throat with false membrane, this application should be used :—

℞	Menthol.	partes ij.
	Camphor.	part. j.

Adults should gargle with fresh Labarraque's solution (50 per 1,000) or oxygenated water of 12-volume strength, diluted with boiling water. In children who cannot gargle, the mouth must be washed out with the same solutions, by means of an india-rubber syringe. Gargling or washing ought to be carried out seven or eight times in the twenty-four hours. The use of the tooth brush must be insisted on, when it can be borne. Either before or after painting the pharynx, there should be instilled into each nostril one teaspoonful of :—

℞	Camphor.	gr. iijs.
	Menthol.	gr. v.
	Resorcin.	gr. xxx.
	Ol. Olivæ Steril.	℥ij.

So long as swallowing is painful, liquid food must be given, gruel, eggs, and milk; but when the sore-throat is well, solid food must be taken: meat, fish, vegetable soups made from fresh or dried vegetables, cakes, jams, eggs, fats, and bread, all without salt. Diuretic "tisanes" must be given freely (uvæ ursi and the like), to which may be added syrup of broom. Evian water is equally good, owing to the very small amount of NaCl contained in it.

The cleansing of the skin, the mouth, the pharynx, and the nasal fossæ must be kept up for at least three weeks. The patient may be allowed up for a few hours each day, and, on the twentieth day, begin to take a hot bath daily, which contains 25 grammes of lysol.

For the treatment of acute post-scarlatinal nephritis, milk is indispensable, the salt-free diet must be given, and also diuretic drinks. This treatment is sufficient to bring about the cure of recent scarlatinal nephritis.

Reviews of Books.

NOTES ON NEW EDITIONS.

Collected Papers on Circulation and Respiration. First Series, chiefly containing Laboratory Researches. By Sir Lauder Brunton, M.D., D.Sc., LL.D. (Edin. and Aberd.), F.R.S., F.R.C.P., Consulting Physician and Late Lecturer on Pharmacology and Therapeutics to St. Bartholomew's Hospital. Pp. xiii + 696. London: Macmillan & Co., Ltd. 7s. 6d. net.

As indicated by its title, this book is a collection of papers, written at various times, either by Sir Lauder Brunton, or by him in conjunction with one of his distinguished assistants, and published in different journals and reports. These papers have been collected and reprinted in response to a general desire that the records of such valuable research work should be in a permanent form, easily accessible. The result is a goodly volume, well printed, moderately priced, containing no less than thirty-eight separate articles, including, by way of an introduction, the Harveian Oration, delivered in 1894. An exceedingly interesting preface, largely biographical, at once arrests the attention of the reader, and a very copious index adds very considerably to the value of the book. The profession owes a debt of gratitude to Sir Lauder Brunton for thus placing within its reach such an interesting and valuable record of a portion of his life's work.

A System of Medicine by many Writers. Edited by Thomas Clifford Allbutt, M.A., LL.D., M.D., D.Sc., F.R.S., and Humphry Davy Rolleston, M.A., M.D., F.R.C.P. Vol. II., Part I. Pp. 1087. London: Macmillan & Co., Ltd. 25s. net.

The second volume of this work, as originally published, has been divided into two parts, of which Part I. contains the continuation of the "Infectious Diseases and the Intoxications," together with an excellent and extended article upon the "General Pathology of Infection," by Professor James Ritchie. It also contains a new article on the "Pathology of the Streptothrix Infections" by Mr. Foulerton, and "An Account of the Bacteriological Relations of Rheumatic Fever" by Dr. Bulloch. The article upon "Tuberculosis" has been revised by Dr. Bosanquet, and an extremely good article on "Food Poisoning" has been added, which comes from the pen of Dr. Batty Shaw. The production of this volume reflects great credit on all concerned.

A System of Medicine by many Writers. Edited by Thomas Clifford Allbutt, M.A., M.DS., D.Sc., F.R.S., etc., and Humphry Davy Rolleston, M.A., M.D., F.R.C.P. Vol. II. Part II. Pp. 1055. London: Macmillan & Co., Ltd. 25s. net.

The present volume has been entirely rearranged, and now contains particulars of all the Tropical Diseases and Animal Parasites; it is, in consequence, practically a complete work on Tropical Medicine. By all those interested in tropical diseases, it will be welcomed as a most valuable book of reference and study. Professor E. A. Minchin has written a special article upon the Protozoa; Mr. F. V. Theobald an excellent article upon Mosquitoes; Mr. Ernest E. Austen an article upon Blood-sucking and other Flies, known or likely to be concerned in the spread of disease; and Mr. R. I. Pocock has contributed an interesting article upon Ticks. Throughout the book, the editors have had the advice and assistance of Sir Patrick Manson.

Elements of Practical Medicine. By Alfred H. Carter, M.D., M.Sc., Fellow of the Royal College of Physicians, London; Professor of Medicine, University of Birmingham; Senior Physician to the Queen's Hospital, Birmingham; Emeritus Professor of Physiology, Queen's College, Birmingham, etc., etc. Ninth edition. Pp. xvi + 614. Crown 8vo. London: H. K. Lewis. 10s. 6d.

We have again to chronicle the issue of a new edition, this time the ninth, of this admirable little work. While the original plan of the work, as an elementary introduction to the study of medicine, has been adhered to, the text has been carefully revised throughout, many sections have been rewritten, and some necessary additions have been made. In the revision of the chapters upon Pathology and Nervous Diseases, the assistance of Dr Stanley Barnes has been secured, and Dr. Douglas Heath has afforded valuable help in revising the section on Diseases of the Skin. To mention Dr. Carter's valuable book is to strongly recommend it, and the knowledge that the new edition has been so thoroughly and ably revised, cannot fail to commend it to earnest students.

Auscultation and Percussion, together with the other Methods of Physical Examination of the Chest. By Samuel Gee, M.D., F.R.C.P., Honorary Physician to H.R.H. the Prince of Wales, and Consulting Physician to St. Bartholomew's Hospital. Fifth edition. Pp. xviii + 325. London: Smith, Elder & Co. 6s.

The appearance of a fifth edition of this scholarly and eminently practical work is conclusive evidence that its popularity, arising from proved worth, is in no way diminished. The book itself, like the author's reputation, is far too firmly established as a scientific authority to allow of any criticism. It has been a trusty friend to innumerable men now qualified, and will prove equally so to all those who will follow in their footsteps. It is a hardy perennial, which "age cannot wither."

The Schott Methods of the Treatment of Chronic Diseases of the Heart, with an Account of the Nauheim Baths, and of the Therapeutic Exercises. By W. Bezly Thorne, M.D., M.R.C.P. Fifth edition. Pp. 140. 42 Illustrations, Plates and Diagrams. London: J. & A. Churchill.

The issue of this the fifth edition of this work, gives Dr. Thorne the opportunity to adduce further evidence of the value of the treatment in aortic regurgitation, as well as in certain conditions, not primarily cardiac, such as Raynaud's disease and Graves' disease. The book will be found of great practical use to all who wish to have full information about this method of treatment, which is now acknowledged to render valuable service in the difficult task of improving the local and general condition of heart cases.

On Retro-Peritoneal Hernia: being the "Arris and Gale" Lectures on the Anatomy and Surgery of the Peritoneal Fossæ delivered at the Royal College of Surgeons of England in 1897. By B. G. A. MOYNIHAN, M.S., F.R.C.S. Second edition. Revised and in part rewritten by the Author and J. F. DOBSON, M.S., F.R.C.S. Pp. vi + 195. 40 Illustrations and 12 plates. Demy 8vo. London: Baillière, Tindall, & Cox. 7s. 6d. net.

THE great advance in anatomical work during the last ten years, and the additional care which has been bestowed upon the reporting of cases, have, of necessity, brought further light upon the subject-matter of these Lectures, and rendered a revision of it inevitable. In preparing this edition Mr. Moynihan has had the able assistance of Mr. J. F. Dobson, so that all work of consequence has passed under review, and the more important cases have been quoted. The value of this edition is enhanced by the excellent plates and illustrations, the addition of some of the latter being due to the pencil of Miss Ethel M. Wright.

Tumours, Innocent and Malignant: Their Clinical Characters and Appropriate Treatment. By J. BLAND-SUTTON, F.R.C.S., Surgeon to and Member of the Cancer Investigation Committee of the Middlesex Hospital, etc. Fourth Edition. With 355 Engravings. London: Cassell & Co., Limited. 21s. net.

THE first edition of this admirable work was published in 1893, and that a fourth edition is already called for is a signal proof of its value, and also of the keen interest taken in the subjects of which it treats.

In the present edition, as might be expected, special attention is paid to cancer research, and an account of the prevailing but irreconcilable opinions as to the origin of the disease, which are held by competent observers, is included. The hypotheses, which have been formed as to the causative agent, are fairly and impartially set forth. New matter, relating to tumours of the ovary and the testicle, and a chapter on heterotopic teeth, as well as 50 new illustrations, involving an increase of more than one hundred pages, have been added.

The illustrations throughout the book are so admirable, and its subject matter so ably dealt with, that the gratitude of the Profession for the distinguished author is fairly earned, and will doubtless be liberally bestowed.

The publishers are entitled to commendation for the manner in which their task has been discharged.

Clinical Lectures on Enlargement of the Prostate. By P. J. FREYER, M.A., M.D., M.CH., Surgeon to King Edward VII.'s Hospital for Officers, and to St. Peter's Hospital, late Examiner in Surgery at the Durham University, Lieut.-Colonel, Indian Medical Service (retired). Third Edition. Demy 8vo. Pp. 154. 54 Illustrations. London: Baillière, Tindall, & Cox. 6s. net.

DR. FREYER'S Lectures on Stricture of the Urethra and Enlargement of the Prostate are already so well known as to require but little comment. The present volume deals only with the causations, symptoms, diagnosis, and treatment of enlargement of the prostate. The author commences with a clear account of the anatomy and relations of the prostate gland, and again emphasises the possibility of its complete extirpation. He describes his operation for total prostatectomy in detail, as well as the after treatment of operation cases, and we note that he does not attach any importance to the attempts which are sometimes made to preserve the prostatic portion of the urethra. The mortality of the operation is given as 7 per cent., and we are further told that, in his last 37 consecutive cases, Dr. Freyer has not had a single fatal result. The book is extremely well illustrated, and many actual cases are described.

Manual of Surgery. By ALEXIS THOMSON, F.R.C.S. Ed., and ALEXANDER MILES, F.R.C.S. Ed. Volume First. Pp. 808. Edinburgh and London: Young J. Pentland.

THIS excellent work is published in two volumes, of which the first deals with general surgery, and the second with regional surgery. The first volume has recently appeared in a second edition, which has been thoroughly revised and enlarged. It contains 313 well-executed illustrations, many of which are new. Some of the sections have been re-written and brought up to date. We do not hesitate to advise those practitioners, who are contemplating entering for any of the higher examinations in surgery held in Edinburgh, to carefully read this book.

Outline of the Diseases of Women. By JOHN PHILLIPS, M.A., M.D., F.R.C.P., Professor of Obstetric Medicine and the Diseases of Women, King's College Hospital, &c. Pp. 282. 121 Illustrations. London: C. Griffin & Co., Ltd. 7s. 6d.

THIS well-known practical manual, written entirely for the use of medical students and junior practitioners, has now reached its fourth edition. It has been thoroughly revised and again brought up to date, and contains a very good account of "Chorion Epithelioma," also two useful appendices, one on the physical signs observed during pregnancy, the other on the advisability of making local (vaginal) examination. We can with confidence recommend the book to those for whom it is especially intended.

First Lines of Midwifery: A Guide to Attendance on Natural Labour for Medical Students and Midwives. By G. ERNEST HERMAN, M.B. (Lond.), F.R.C.P., Consulting Obstetric Physician to the London Hospital, etc., etc. Pp. 222. London: Cassell & Co., Ltd.

WE welcome a new edition of Dr. Herman's well known book on the "First Lines of Midwifery." The whole of the text of the present volume has been revised, and, in a few places, added to. There are also one or two additional illustrations. As the work is so much used by midwives, Dr. Herman has wisely included the set of rules published by the Central Midwives Board for the use of midwives themselves.

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Notes by the Way.

Food Fads.

The diet reformer is abroad in these days, and every month witnesses the birth of a new food fad. We have the Fletcherites, who, so far from not giving two bites to a cherry, insist on thirty-two to a mashed potato, and who even profess to chew their milk and other liquid food ; the followers of Chittenden, who weigh their food and count the morsels, and would seem, like Sanctorius, to pass the greater part of their lives in a balance ; the devotees of Dewey, who teaches that the secret of a healthy life is to eat no breakfast ; the followers of Salisbury with their raw minced beef and hot water ; those whose chief aim in life is to wage war on uric acid, which the physiologists now tell us is a "bogey" ; Mr. Bernard Shaw with his grape nuts ; Mr. Eustace Miles, with his "rissoles with curry flavour," his "rice cutlets," and his "vegetable roast" ; Dr. Haddon with his apples ; others with their salads, their wholemeal bread, or their patent foods of one kind or another, and many more. Then there is Professor Metchnikoff, who says that if we wish to rival the patriarchs in longevity we must kill all the "wild" microbes in our intestinal tract by drinking his special elixir, Bulgarian sour milk ; some who have tasted it have said that they would prefer a short life to length of days saddened by frequent draughts of the genuine *Kisselo Mleko.*

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A Diet of Pills and Tabloids.

The great chemist, whose loss science now mourns, Marcelin Berthelot, not long ago confided to an English journalist his belief that the day might not be far distant when chemistry would effect the economical manufacture of foods. He went on to say that he had devoted a great deal of time and thought to the matter, and had come to the conclusion that the alimentary problem is essentially a chemical one, and that when energy can be obtained economically, it will not be long before it is solved. Rapt into future times he prophesied that foods would then be manufactured with carbon obtained from carbonic acid, with hydrogen extracted from water, and with oxygen and nitrogen taken from the air. "Corn-growing and cattle-raising," he declared, "will then suffer the same fate as the cultivation of madder has done in our day, and the world will be independent of those bad seasons which put the farmers in despair. The world will then . . . " Here, apparently, the vision faded *in tenues auras*, and the world still awaits the revelation of the great secret for which Berthelot perhaps thought it was not yet prepared. Still, more daring scientific prophets profess to look forward to a time when mankind will subsist on pills and tabloids in place of the more substantial viands which are still human nature's daily food. A still more advanced ideal is represented by the Martian race, described by Mr. H. G. Wells in his *War of the Worlds*, who dispense with a stomach!

Professor Ray Lankester thinks that Berthelot's forecast reflects what will really happen. Regular meals he regards as the bane of modern life. Tabloids and chemical essences would offer less temptation to the pleasures of the table. The goal to which we are moving is a time when chemistry will supply our food, and the sun and the terrestrial reservoir will supply our heat, and consequently our mechanical force. "The countryside will then be planted with trees and flowers, and we will no longer be obliged to slay cattle and subsist on their flesh." Against this may be set the opinion of Sir William Crookes, who unkindly calls this kind of thing one of the platitudes of pseudo-science; and that of Sir William Ramsay, who considers Berthelot's views altogether illusory. Chemistry, he thinks, may be able to fabricate any given article of food for human consumption. With albumen it is not impossible that a chemist of the future might compound a passable steak or chop; "but at what a cost, what a waste of skill and labour!" According to Sir William Ramsay, it is an economic fallacy to suppose that such an ingenious concoction could ever take the place of plants and animals produced by the synthetical action of sunshine. To ourselves Berthelot's views recall the philosophers of Laputa who tried to extract sunbeams from cucumbers. Although men of science are sometimes dreamers of dreams as unsubstantial as those of poets, we can scarcely believe that the great French chemist was wholly in earnest.

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The Fruits of "Fletching."

We used to hear much about "banting"; what is perhaps for the moment the most fashionable food fad is called "fletching." It is playfully described by Mr. Dooley as follows: "The latest thing that Carney has took up to make th' fight again' Nature is called Fletching. Did ye iver hear iv it? Well, they'se a lad be th' name iv Fletcher who thinks so much iv his stomach that he w'ont use it, an' he tells Carney that if he'll ate on'y wan or two mouthfuls at ivry meal an' thurly chew thim, he will ivinchooly be no more than skin an' bones an' very handsome to look at. In four weeks a man who Fletches will lose forty pounds an' all his friends. Th' idee is that ye mumble ye'er food f'r tin minyits with a watch in front iv ye." Mr. Dooley adds the reflection: "Man is foolish that thries to be too kind to his stomach, annyhow. Fletcher's idee is th' human stomach is a kind iv Little Lord Fauntleroy. If ye give it much to do it will pine away." It has been said that men dig their graves with their teeth. Mr. Fletcher's disciples do not perhaps dig their graves in that way, but, on the other hand, they reverse the maxim of Harpagon's serving man, and, instead of eating to live, live to eat. Though this is not done out of gluttony, it is a waste of time and energy. And what are its fruits? If we are to believe Dr. F. G. Shattuck, of Boston, they are at best of somewhat doubtful quality. He has been brought personally in contact with some of those responsible for the initiation of the system. Mr. Fletcher's son-in-law, Dr. Van Someren, who, according to Dr. Shattuck, used to be a rather stout and good-natured gentleman, has become cadaverous-looking, although he is satisfied that the new system is effectual in enabling him to maintain his metabolic equilibrium. While in Europe Dr. Shattuck met Dr. Sandwith, of Cairo, who told him that he not infrequently had the opportunity of seeing patients who had been under the Fletcher system, and the main thing he has to do with them is to encourage them to eat more and so build them up to their normal condition of health. We give these statements as we find them in the *Medical News* of August 5, 1905, leaving the responsibility for them to Dr. Shattuck. But it is a matter of common observation that the appearance of dietetic cranks is not such as to serve as a good advertisement of their system. Of course a large amount of muscular work can be done on a vegetarian diet; one need look no further for proof than to the Scottish farm labourer with his porridge, and the Irish peasant with his potatoes. With brain workers the case is different; the literary output of vegetarianism is in large measure of the quality of those cabbage-inspired writings which Herbert Spencer destroyed when he was compelled to return to the fleshpots.

* * *

The Ethics of Eating.

THE truth is that faddism in feeding, whether of the high scientific or the austerely ascetic variety, leaves out of account one of the principal elements in a truly physiological diet. Pawlow, who has done so much to elucidate the process of digestion, has shown that an essential part is played by what he calls "appetite juice." "A good appetitite in eating," he says "is equivalent from the outset to a vigorous secretion of the strongest juice; where there is no appetite, this juice is also absent. To restore appetite to a man means to secure him a large stock of gastric juice wherewith to begin the digestion of the meal." The secretion of the juice is not stimulated by food which one finds no pleasure in eating; the digestion of such things is long and laborious. Food, eaten without relish and without appetite, however nutritious it may be in a theoretical sense, simply cumbers the stomach without serving the purpose of food. The moral of Pawlow's teaching is that in this vale of tears we cannot live on chemical formulæ. The absolute quantity of foodstuffs taken, and the number of calories these quantities represent, do not make up the whole food of man. As Carl Voit puts it, we can take five glasses and put into them enough food to represent the average quantity eaten daily by a robust man, that is, 3,000 grams of water, 118 grams of albumen, 56 grams of fat, 500 grams of starch, and 20 grams of mineral elements. But if we mix these together and offer them even to a very hungry man, he would simply refuse to eat the mess. And if, overcoming his natural repulsion, he did eat it, he would starve. Something more than the crude foodstuffs is needed. White of egg, if given alone, needs a long time for its digestion, whereas a broth or meat extract, which is of no direct value, is useful in preparing the way for more nourishing food by exciting the secretion of gastric juice. For this reason Pawlow has even a good word to say for the much-abused *apéritif*. Another part of what has been called the ethics of eating, which is too often lost sight of, is that the mind should be emptied of the cares of the day when we sit down to table. It is a mistake to eat, as Napoleon did, taking with indifference whatever comes in our way and hurrying through the act as a wearisome necessity. That way lies dyspepsia, with all its attendant miseries.

* * * * *

Common Sense in Diet.

WE are glad to note that the common sense view of the diet question is also taken by a great French authority, Dr. Armand Gautier. Dealing with the subject in the *Revue Scientifique* of March 16, he says that, in the matter of food, we have advanced from prudence to excessive caution, and from this to suspicion of everything. We are afraid of meat, on account of its supposed toxins; of broth, because of its nitrogenous waste matters; of vegetables, because they dilate the stomach; even of watercress, radishes, and strawberries, because of microzoa and bacteria. Fear of tuberculosis leads us to taboo milk and butter; bread is believed to charge the blood with acids; and water carries in it the germs of typhoid fever. As for wine, it is anathema maranatha. As M. Gautier says, "Physiologists of delicate stomach, and doctors and philanthropists preoccupied with the evils of alcoholism, all those who cannot digest wine (for there really are certain stomachs which cannot tolerate it), lastly, some physicians who had the curious idea of drawing their conclusions from direct injections of alcohol into the veins or under the skin: all these theoricians declare that alcohol, and by extension, wine, are simply poisons." Hence wine has fallen out of favour. This, according to our author, is a great mistake. Dr. Gautier enjoins us to steer clear of all such exaggerations. Let us, he says, boil our water in time of epidemic, let us have our beefsteak sufficiently done, and our milk cooked, and we may sleep in peace. A plate of light soup, a slice of roast beef done to a turn, a small glass of claret or even Burgundy, never killed anyone.

Let us eat regularly, as appetite prompts, viands that have from all time been recognised as harmless; let us only use some extra precautions in the choice and cooking of our water and food in times of epidemic. If bread, meat and wine do slowly poison us, we may at least feel assured that the moderate use of these things will shorten our lives less than the fear engendered by all those extra scientific exaggerations which overshadow our days. It is neither meat, nor soup, nor wine, nor condiments, nor coffee that gradually poison us; it is the abuse of them. We should trust our own sensations, provided they are natural, and not artificially produced, rather than tables of analyses. The whole philosophy of diet lies in following this simple law, which is consecrated by the immemorial experience of the whole human race, and in avoiding excess. This is embodied in the precept of the School of Salerno: —"Pone gulæ metas ut sit tibi longior ætas."

* * * * *

Natural Death.

How many people die a natural death? If we are to believe Metchnikoff, very few indeed. A natural death is that which results necessarily from the organisation of a being, and not from accident. Although in common speech, death by illness is called natural, illness is avoidable, and is not an inevitable result of the operation of the forces which make up life. Although people die around us daily and hourly, so that in the midst of life we are in death, it is remarkable how little attention science has given to death. It can easily be understood why doctors have not made a particular study of the subject. Their business being to prolong life, they look upon death as the supreme expression of the limitation of their art, and no fighter cares to think much of a defeat. Nor does it appear that the study of natural death has any special lesson for them, for until they learn how to keep off old age, they can do nothing to prevent the decay, which is the consequence of the wearing out of the machinery of life.

Metchnikoff thinks it probable that both sleep and its brother, death, are the results of an auto-intoxication of the nervous centres by poisons produced by fatigue. The exhaustion thus engendered induces an instinctive need of rest, and this, in an infinitely greater degree, may manifest itself in an instinctive longing for death. As an example of this instinctive desire for the last sleep, Metchnikoff quotes the following story from Brillat-Savarin, the celebrated author of the *Physiologie du Goût*:—

"I had a great-aunt ninety-three years old, who was dying. Although for some time confined to her bed, she had retained all her faculties, and her condition was betrayed only by her loss of appetite and the weakening of her voice. She had always shown a fondness for me, and I was near her bed, affectionately ready to wait on her, which did not prevent my watching her with the philosophical eye I have ever had for the things and events surrounding me. 'Are you there, nephew?' she asked in a scarcely audible voice. 'Yes, Aunt; I am here at your service, and I think you would do well to take a little good old wine.' 'Give it me, *mon ami*; one can always swallow liquid.' I hastened; raising her gently, I made her take half a glass of my best wine. She brightened for a moment, and looking at me with eyes that had once been very fine, 'Thank you,' she said, 'for this last favour; if *ever you reach my age, you will find that death becomes a need just like sleep*.'"

As a consoling thought, Metchnikoff suggests that the instinct of natural death is, in all probability, accompanied by "as peaceful and pleasant a sensation as can be conceived." Those who have stood by many deathbeds say that, in the majority of cases, the soul parts from the body without fear or suffering. Death, whether natural or accidental, comes indeed to most as a friend, and they,

Craving nought nor fearing,
Drift on through slumber to a dream and through a dream to death.

◆

Practical Notes.

THE POSTERIOR NERVE-ROOT GANGLIA.—The posterior nerve-root ganglia are derived from the epiblastic *neural crest*, which develops along the dorsal aspect of the primitive spinal cord. Special local enlargements of this neural crest appear opposite the middle of each pair of protovertebræ, and then their attachment to the spinal cord becomes entirely lost. In these islands of epiblast, many *neuroblasts* appear, and these form the spinal ganglia. From the contained neuroblasts, the cells of the spinal ganglia are developed. From each nerve cell two processes arise, one of which grows away towards the periphery and ends, as a rule, in a sense organ there, the other grows into the spinal cord, producing there the exogenous nerve-fibres. These two processes, which have developed from the bipolar nerve-cell, later become blended, so that a unipolar nerve-cell replaces the original bipolar nerve-cell, and consequently a **T**-shaped junction is produced. The central fibres, which then arise in the posterior nerve-root ganglion cells, form the posterior nerve-roots of the spinal cord. They then pass into the posterior root zone of the cord, where they bifurcate, one branch, as a rule, travelling up the cord, and the other travelling for a short way downwards. From the chief fibre and its branches collateral fibres arise, which pass at frequent intervals into the grey matter, and there end by arborising around the small nerve-cells in the neighbourhood of the posterior grey cornu, and around the cells of Clarke's column, which are found in the dorsal region of the spinal cord. The main fibres pass into the postero-lateral column of Burdach, and higher in the cord, they pass into the postero-mesial column of Goll, until they arrive at the medulla oblongata, where the medullated nerve fibres end by terminal arborisations around the cells of the nucleus cuneatus and nucleus gracilis, whence new fibres arise which pass to the cerebrum and cerebellum. Most of the smaller fibres enter and ascend the cord in the marginal bundle of Lissauer, which lies external to the posterior grey cornu. Some of the fibres from the posterior nerve-root pass directly into the posterior grey cornu, and arborise around cells at its base.

SECTION OF THE POSTERIOR NERVE-ROOTS.—It has been found, experimentally, that cutting the posterior nerve-roots in an animal between the spinal ganglion and the cord, leaves the peripheral part of the nerve healthy, but degeneration occurs in that part of the nerve between the cut and the spinal cord. This is due to the fact that the nerve fibres are severed from the cells, from which they originally grew, and the cells exercise a trophic influence over the fibres.

If several posterior nerve-roots are cut between the posterior spiral ganglia and the cord, the degeneration which occurs may be readily traced. Directly below the points of entry of the nerve-roots, it will be found that some nerve-fibres in the cord have degenerated downwards, whereas, immediately above, there will be an ascending degeneration in the nerve-fibres which constitute the column of Burdach, higher up the cord still, there is degeneration of the fibres in the column of Goll, and if the nerve-roots, which have been cut, happened to be in the lower cervical region of the cord, there would also be an ascending degeneration in the marginal column of Lissauer.

LOCOMOTOR ATAXIA.—There is much divergence of opinion as to the position and character of the initial process in tabes dorsalis, or posterior spinal sclerosis, but of the causes syphilis is undoubtedly the most important. Of 300 cases of tabes dorsalis, occurring in his private practice, Erb states that 89 per cent. had had syphilis, and Moebius says, "The longer I reflect upon it, the more firmly I believe that tabes never originates without syphilis." In advanced cases of tabes, Mott has found some alteration in the nerve-cells and the spinal ganglia with degeneration of the dorsal nerve roots, also a degeneration of the exogenous nerve fibres of the spinal cord, which develop from the posterior nerve-root ganglion cells. Obersteiner and Redlich have laid stress on the evidence of inflammation, which they have observed in the pia mater, which covers the dorsal aspect of the spinal cord, and consequently involves the posterior nerve-root fibres as they pass forward through it. In a similar manner, the nerve cells, which exist in the Gasserian ganglion, have been found to be degenerated, and degenerative processes have also been found to have occurred in the various divisions of the fifth cranial nerve.

TREATMENT OF TABES.—If the syphilis, which has caused the disease, was acquired as recently as within two or three years, benefit arises to the patient by a thorough course of mercury and increasing doses of potassium iodide. More recently, it has been suggested by some of the French physicians in the earliest stages of tabes, even though the syphilis was acquired some years before, that the patients should be put through a thorough course of mercurial inunctions, and that fairly large doses of potassium iodide should be administered internally. Dr. Burney Yeo says, however, that, in the later stages of the disease, anti-syphilitic treatment can do no good, but will probably cause harm, and this line of treatment is also contra-indicated in those cases, in which optic-nerve atrophy occurs as an early symptom, for mercury has been found to hasten and not retard this process. Morphia should be given for the severe paroxysms of pain. For the spasms, associated with laryngeal crisis, cocaine may be applied locally, or a little chloroform inhaled.

When ataxia sets in, and the patient loses control of some of his voluntary movements, there is no doubt that Fraenkel's method of re-education of these muscles is of especial benefit. By this method, the patient is taught to perform simple movements, and when he has acquired these, he is then taught more complex ones, and so, in this way, the power of walking is often very considerably improved.

Professor Alfred Fournier[1] says: "The usual ointment for inunction is the double mercurial ointment, called Neapolitan ointment, which is composed of equal parts of mercury and lard. Lanoline may be substituted for the latter, and is said to penetrate the skin better than lard. Mercurial soaps are recommended by some, and are said to be less dirty than inunctions with a fatty basis, and also less irritable. But they have the great disadvantage of requiring a considerable time for absorption, so that mercurial soaps and other preparations, such as the oleate of mercury and calomel, have not succeeded in replacing the old mercurial ointment. The average dose for an adult is a drachm, and this may be increased to 1½ or 2 drachms, according to the tolerance of the patient."

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[1] *The Treatment of Syphilis.*

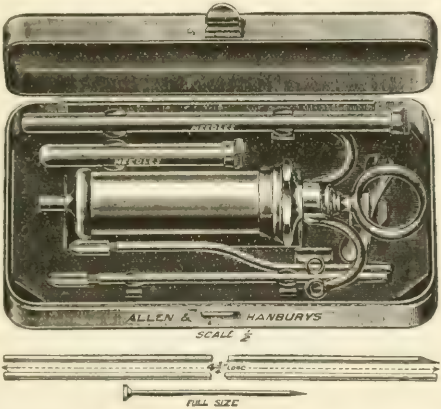
GASTRIC CRISES.—When these occur in tabes, the patient experiences severe pain in the epigastrium followed by vomiting and an increased secretion of hyperacid gastric juice; vomiting, however, is not a constant symptom. The attacks vary in their intensity from time to time. The patient may also suffer from other crises in other parts of the body such as the larynx, kidneys, rectum, and urethra. It is important to remember that, as these crises occur in the pre-ataxic stage of tabes, the association may be readily overlooked, unless the ocular symptoms are noted, and the knee-jerks are found to be absent. Severe and sudden gastralgic attacks occur in association with gastric ulcer, and they may occur also as a manifestation of a functional neurosis quite independent of any local organic disease. In this latter condition, the severe gastrodynia, or gastralgia, is usually associated with other nervous symptoms, which are much more frequent in women than in men. Sir William Whitla says the best treatment for gastric crises is by a smart counter-irritant applied to the epigastrium, morphia hypodermically, followed later by gastric sedatives, such as Antipyrin (10 grains), Chloroform (5 minims), Ether (5 minims), Sodium Bicarbonate (15 grains), Oil of Peppermint (5 minims), Oil of Cajuput (4 minims), Tincture of Belladonna (15 minims), Dilute Hydrocyanic Acid (3 or 4 minims), which may be tried from time to time.

Sometimes patients, who suffer from gastric crises, have co-existent dyspepsia, this must be treated by careful regulation of the diet, and by the administration of some preparation of bismuth and an alkali, together with dilute hydrocyanic acid. If the vomiting is persistent, cerium oxalate may be prescribed. It is most important that the bowels should be kept relieved every day, and, with this end in view, simple aperients, such as the compound liquorice powder, a teaspoonful or two of the confection of senna, or a little cascara sagrada may be administered.

Preparations, Inventions, etc.

A NEW ALL-METAL SYRINGE FOR EUCAINE.

(Messrs. Allen and Hanburys, Ltd., Wigmore Street, W.)



The syringe, as shown above, is a modified pattern of the instrument invented by Mr. A. E. J. Barker, F.R.C.S., and has been made by Messrs. Allen and Hanburys according to the suggestion of Major Porter. It is of 20 cc. capacity, and is constructed throughout of hard metal, so that it can be readily sterilised without getting out of order.

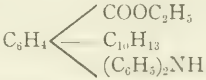
The metal piston is so accurately fitted to the barrel that a little sterilised oil is required to lubricate it before use. It is graduated and fitted with a revolving stop, and there is no possibility of fluid getting behind it when injecting into dense tissues—an advantage which will at once commend itself. The needles for use with this syringe are larger than those formerly employed, and the long cannulæ correspondingly so. The syringe and fittings are all nickel-plated and are fitted into an aseptic case at a cost of 45*s.*

ARHOVIN (DR. HOROWITZ).

(Messrs. Reitmeyer & Co., 63, Crutched Friars, E.C.)

Arhovin, prepared in the Chemical Institute of Dr. Horowitz, Berlin, is a product of diphenylamin and the esterified thymolbenzoic acid, and is a yellowish liquid of aromatic odour and slightly cool-burning taste. It is almost insoluble in water, but readily soluble in alcohol, ether, chloroform, and fatty oils.

Its formula is



$$C_6H_4\left\langle\begin{array}{l}COOC_2H_5\\C_{10}H_{13}\\(C_6H_5)_2NH\end{array}\right.$$

Its use internally is indicated in acute, subacute, and chronic gonorrhœa of the anterior and posterior urethra, prostatitis, cystitis, pyelitis, and also, externally, in acute, subacute, and chronic gonorrhœa of the urethra in both sexes; cystitis, colsitis, fluor albus, adenitis Bartholiniana, endometritis, osphoritis, proctitis.

Arhovin is supplied to the profession in 1-oz. bottles, in boxes of capsules, bougies, and globules.

IODALBIN.

(Messrs. Parke, Davis & Co., Queen Victoria Street, E.C.)

This is an iodo-proteid compound containing 21–5 per cent. of iodine, and is in the form of an almost tasteless powder, insoluble in water, acids, or alcohol, but readily soluble in alkaline solutions. When administered it passes unchanged through the stomach, and is gradually absorbed in the intestine, thus avoiding the gastric irritation that the alkaline iodides are likely to excite, and also ensuring a milder systemic effect.

Iodalbin has been found to produce the typical alterative effect of the organic iodides without their disadvantages; it may, therefore, be prescribed in preference to these in cases in which such treatment is indicated. It is supplied in bottles of one ounce, and in capsules (5 grains each) in bottles containing 25 and 100.

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